SERIES 3, Vol. 14, No. 9 SEPTEMBER, 1931

SEP 22 1931

AMERICAN JOURNAL OF OPHTHALMOLOGY

Medical Lib.

CONTENTS

Original Papers Page

Contribution to the anatomy of congenital ectropium uveæ (flocculi iridis). Prof. Dr. Adalbert Fuchs ... 865
An internist's inquiry regarding the etiologic factors of uveitis. Willis S. Lemon ... 869
The pathology of papilledema. W. E. Fry ... 874
Xeroderma pigmentosum. F. B. Blackmar ... 884
Deep orbital injury by stick of wood: exophthalmos and optic atrophy following. H. L. Hilgartner and H. L. Hilgartner, Jr. ... 886
Parathyroid cataract. M. E. Marcove ... 887
Variations of the normal blind spot with special reference to the formation of a diagnostic scale. Hazel A. Wentworth ... 889
Ophthalmology in aviation medicine. William F. Holzer ... 905
Epinephrin in progressive myopia. Leo L. Mayer ... 908
Annular scleritis. Charles W. Tooker ... 911
An improved double rotary prism. Wendell L. Hughes ... 915
The possible relation of strong lights in the motion picture industry to lens changes. Lloyd Mills ... 917
The relation of the basal metabolic rate to progressive axial myopia. Louis Bothman 918
Nickel sensitization from white gold spectacle frames. A. W. McAlester, Jr., and A. W. McAlester, Third ... 925
Ocular symptomatology in acute poliomyelitis. Henry M. Emmons ... 927
Ocular disease and the vermiform appendix. Edward Bellamy Gresser ... 929
Senile macular changes of the retina associated with focal infection. Hunter W. Scarlett ... 932
Concerning simple glaucoma. Harry S. Gradle ... 936
A glaucoma study. Michael Goldenburg ... 944
Evaluation of pseudo-isochromatic tests for the detection of color-blindness. J. Ellis Jennings ... 952

Notes, Cases, Instruments

Diplopia corrected by high degree prisms. A. F. Luhr ... 959
Enucleation snare. C. W. Banner ... 959

Society Proceedings

Nashville, Royal, Chicago ... 961

Editorials

The Oxford Ophthalmological Congress ... 967
Problems for the Ophthalmologist ... 968

Book Notices ... 970

Abstract Department ... 972

News Items ... 998

For complete table of contents see advertising page V

Copyright, 1931, Ophthalmic Publishing Company, 508 Metropolitan Building, Saint Louis, Missouri

Subscription twelve dollars yearly. Single number, one dollar twenty-five cents.

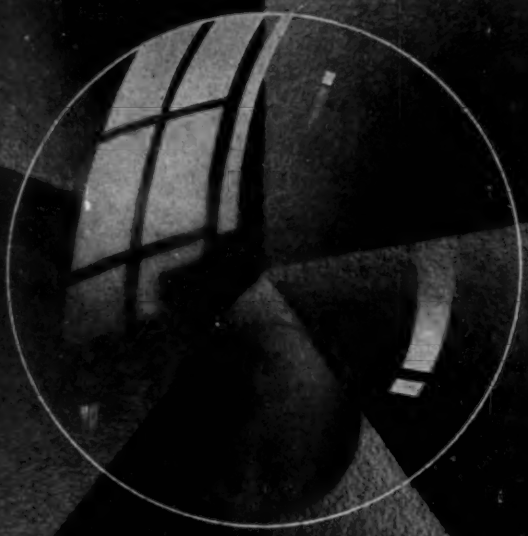
PUBLISHED MONTHLY BY THE OPHTHALMIC PUBLISHING COMPANY

BUSINESS OFFICE: 508 METROPOLITAN BUILDING, SAINT LOUIS, MISSOURI

EDITORIAL OFFICE: 524 METROPOLITAN BUILDING, SAINT LOUIS, MISSOURI

Entered as second class matter at the post office at Menasha, Wisconsin

A NEW STAR IN THE OPTICAL FIRMAMENT...



Announcing

ORTHOGON "D"

a sensational achievement

Read these remarkable facts:

1. Lowest price ever offered for a corrected color-free Bifocal.
2. Front surface segment of 20 mm, the most useful size.
3. Nokrome segment—no color aberration.
4. Astigmatically corrected in all powers.
5. Unsurpassed invisibility of segment.
6. Finest quality manufacture.

Orthogon "D" has the features that appeal instantly to the practitioner and to the public. It meets the quality standards of the most particular practitioner. Its price is well within that which the majority of your patients are willing to pay. This assures its wide usage.

Riggs will gladly send you complete information without cost or obligation. Just check coupon below and send to Riggs Optical Company, Box 3364, Merchandise Mart, Chicago, or to your nearest Riggs office.

( ) Send me Orthogon "D" descriptive booklet.

( ) Send me Orthogon "D" price list.

( ) Send Riggs Representative to explain the Orthogon "D" features.

My Name.....

My Address.....

RIGGS OPTICAL COMPANY

There's a Riggs Office Conveniently Near You.

AMERICAN JOURNAL OF OPHTHALMOLOGY
Volume 14 September, 1931 Number 9

CONTRIBUTION TO THE ANATOMY OF CONGENITAL ECTROPIUM UVEÆ (FLOCCULI IRIDIS)

PROF. DR. ADALBERT FUCHS
VIENNA, AUSTRIA

TRANSLATED BY BERTHA KLIEN-MONCREIFF, M.D.

Two cases of this interesting formation are described clinically and histologically with drawings showing it to consist of an abundant growth of large pigment epithelial cells giving rise to the formation of folds and figures of spiral form. From the Ophthalmic Department of the Allgemeine Policlinic in Vienna (Prof. Dr. A. Fuchs). Translator is Assistant Professor of Ophthalmology, Rush Medical College, Chicago.

In the literature subsequent to 1920 one finds various descriptions of a congenital anomaly, which was first described in the year 1869 by Colsmann, as papilloma iridis, and then in 1873 by E. C. Holmes of Chicago, who even at that time likened it to the pigment proliferation which one finds especially often in horses.

Fig. 1.

Fig. 2.

Subsequently in Hirschberg's Clinic[1], the designation Ektropium Uveae Congenitum was employed, which subsequently was used for a long time, until Stahli in 1920 redescribed the anomaly and again referred to its similarity to the "Traubenkörner" of horses, applying the term Flocculus formation.

Clinically this condition is well known, and is not of such especially rare occurrence. It may be described as consisting of dark brown wart- or tuft-like excrescences of the pigment epithelium, which partly project into the pupil, and partly are bent forward, resting on the anterior surface of the iris. Usually they are symmetrically situated, and are best developed in the upper half of the pupil. Their connection with the pigment epithelium is easily seen with the slit-lamp. These pigmented flocculi may become separated from the pupillary margin, fall into the anterior chamber and remain floating freely in the aqueous, or may disappear from time to time behind the limbus, reappearing with changing positions of the head. Occasionally they give rise to cyst formation, as E. Fuchs[2] has described. Schüller[3] reports a case in which such a free pigment body in the anterior chamber was observed by the patient himself, and mistaken for a foreign body.

In spite of the frequency of ectropium uveae congenitum, which has been amply described clinically, histological studies in man have been few, although there are numerous descriptions of it in

domestic animals. Consequently I shall give the anatomy of this condition in the human eye in two cases.

Case 2459. History: A man 53 years of age came to me with the complaint of failing vision of the left eye of several months' duration. A sarcoma of the choroid was found to be the cause of the trouble. Both irides exhibited a few excrescences of the pigment epithelium at the upper pupillary margin. The histological examination revealed multiple excrescences of the pigment epithelium around the pupil, some of them very well developed. Most of them were deeply serrated in such a way that in cross sections the transition of the posterior layer of the epithelium into the anterior layer was not a simple turning, but at the turning point there were indentations from the front or back (Fig. 1-3).

Fig. 3.

Fig. 4.

A few of these excrescences were connected with the epithelium by only a thin band, which accounts for the appearance in the sections of apparent isolation or cutting off from the epithelium of some of the large leaf-like parts within the pupil. Fig. 4 shows an excrescence with marked multiple serrations. These excrescences projected partly straight forward into the anterior chamber, and were partly bent over so as to rest on the anterior surface of the iris (Fig. 2), without, however, having a direct connection with it. In fact, in one place (Fig. 3) it was distinctly discernible that the connection of the excrescences did not reach further forward than the pupillary margin of the sphincter muscle, and they had no connection with the anterior border layer, which begins at this point. The larger excrescences were so large that they could easily be seen in the sections as black points, with the unaided eye. The diameters of the larger flocculi were 0.29 mm., 0.39 mm., respectively, and the largest was 0.46 mm.

Fig. 5.

Case 2584. The second eye was that of a negro from America, in sections of which these flocculi occurred. This eye was very markedly pigmented, but not so deeply, however, as negro eyes from Central Africa. The anterior border layer was densely filled with large clumps of pigment. The pigment epithelium, knotty, thickened and folded, projected 0.3 mm. into the pupil (Fig. 5), at first lining only the free margin of the pupil, and thence extending centralward away from the iris. Tangential sections through the pupil may produce formations which simulate the findings in this case. In such tangential sections, the

Fig. 6.

pupillary pigment seam also appears markedly enlarged, on account of the oblique direction of the section. Thickenings resembling flocculi may be produced also by a somewhat irregular pupillary margin of the pigment epithelium. One glance at the contralateral side and at the width of the pupil shows this error. Fig. 6 shows a very slight ectropium uveae in a tangential section. One recognizes the direction of the section by some isolated folds the cross sections of which are seen near the pupillary margin on both sides within the narrow pupil. The ectropium is indicated by the pigment epithelium resting on the anterior surface of the iris.

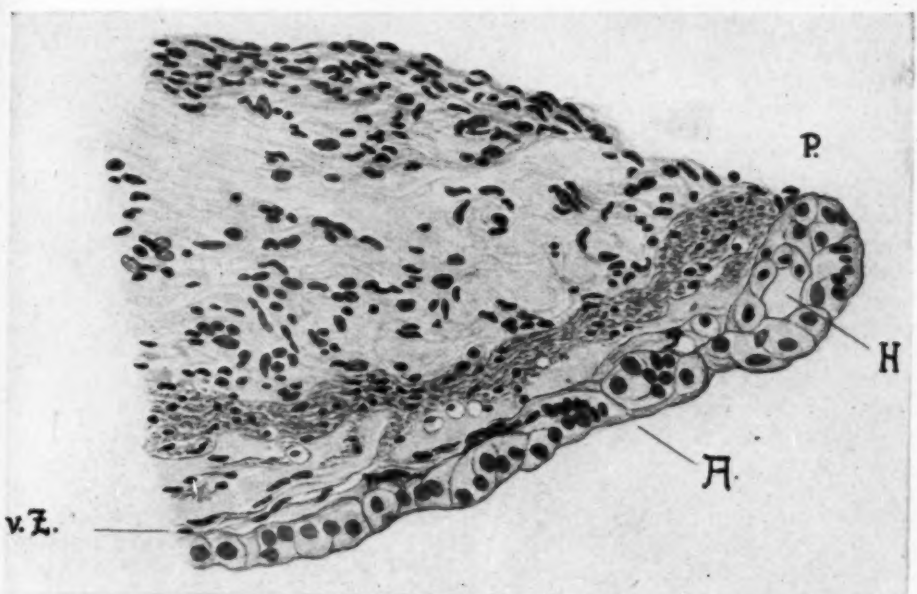


Fig. 7 (Fuchs). Section from albinotic eye (X 250).

In order to recognize the finer histological structure of the flocculi, I studied some depigmented sections of the first case. For purposes of comparison I want to describe first the pupillary margin of an albinotic eye, since the normal conditions at the turning of the inner epithelial layer into the outer layer are well seen in these cases.

The albinotic eye (Fig. 7) does not show a trace of pigment. At A, 0.22 mm. from the anterior margin of the pupil, the cells of the anterior layer of the pigment epithelium (V.Z), which are more flat, spindle shaped and attenuated in the periphery, forming the dilator muscle, become rounded and cuboidal. This layer resembles more and more the posterior layer of cells as it turns at the pupillary margin (P) and merges into the posterior layer. A little cavum (H), about the size of two epithelial cells, enclosed by the two layers of epithelium, is visible in this part of the pupillary margin. It is not situated directly behind the turning point, and corresponds probably to von Szily's sinus of the embryonic eye. How far it extends within the pupillary margin, I cannot tell, as I do not possess the entire series.

The depigmented sections (Fig. 8) of the first case of ectropium uveae congenitum show very well the various kinds of cells. No cavum is seen between the two epithelial layers; the cells which form the excrescences are all relatively large, larger than those adjoining them immediately behind. The anterior and posterior layers of the pigment epithelium are about equally thick within the pupillary margin in a normal eye, and the cells are of about equal size (Fig. 7, to the right of A). In our case the anterior layer of cells is very low, and the cells are considerably smaller than those

of the posterior layer, both layers of cells becoming equal in size only just before the epithelium thickens, forming the flocculus (A, Fig. 8). This may explain why in many of the sections from the first case (Figs. 1-3) and also from the second case (Fig. 5) the pigment margin in normal eyes, I found nearly always values between 50 and 70 microns, the width of the pupil being of no importance for the thickness of the epithelium.

Apparently, therefore, ectropium uveae congenitum consists of an abun-

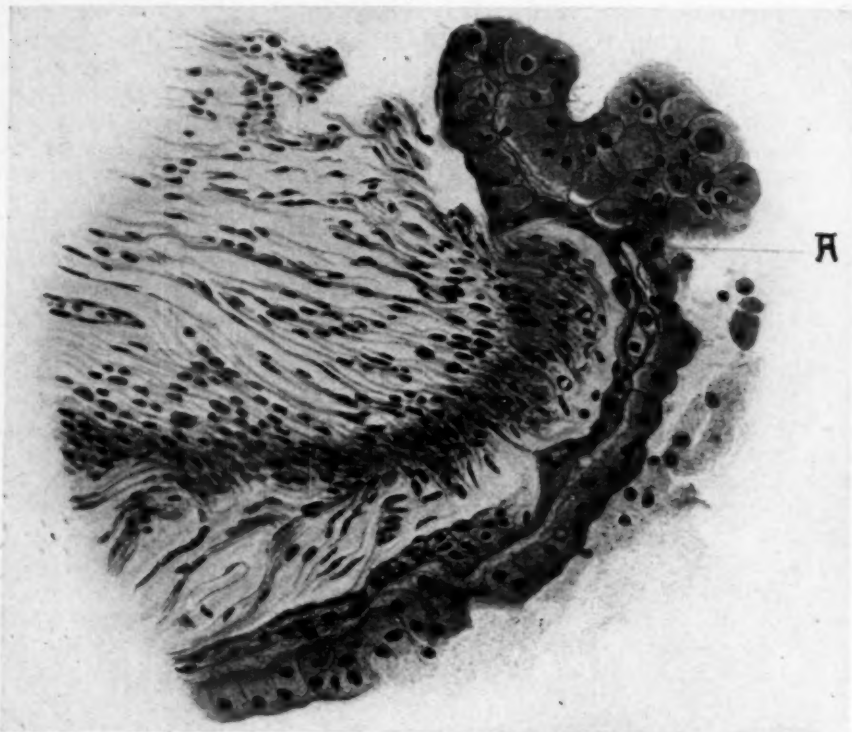


Fig. 8 (Fuchs). Depigmented section; case 2459 (× 250).

epithelium (both layers together) was abnormally thin at or somewhat peripheral to the pupillary margin. I found the thickness of the layers different in different places, for example 22 microns (Fig. 3), 24 microns, and 34 microns (Fig. 1 and Fig. 5). In measuring the thickness of the two epithelial layers together immediately behind the pupillary dant growth of large pigment epithelial cells, which give rise to the formation of folds and figures of spiral form on account of their abundant growth, with a thinning of the epithelium near the zone of reflection, where the epithelium turns from the posterior surface of the iris to the free margin of the iris.

Allgemeine Policlinic.

References

[1] Hirschberg. Klin. M. f. Augenh., v. 65, p. 349.
[2] Fuchs, E. Graefes Archiv., v. 15, p. 7.
[3] Schüller. Klin. M. f. Augenh., v. 74, p. 154.
[4] Cosmettatos. Ann. d'ocul., v. 161, p. 761.
[5] Colden. Klin. M. f. Augenh., v. 71, p. 15.
[6] Greeff. Graefes Archiv., v. 105, p. 134.

AN INTERNIST'S INQUIRY REGARDING THE ETIOLOGIC FACTORS OF UVEITIS

WILLIS S. LEMON, M.B. (Tor.)
ROCHESTER, MINNESOTA

The criteria by which the internist determines the etiologic factors of uveitis are discussed. The internist must know the characteristics of the lesion in the eye and possible changes in other organs that might be causative. Tuberculosis is recognized as one cause of uveitis. The internist thinks it is almost certainly secondary to tuberculosis elsewhere, from which bacteria reach the eye or from which an allergic reaction is set up in the eye. Clinical and nonclinical tuberculosis and the diagnostic features are considered. Diagnosis is not to be made from symptoms alone but from exact methods of examination. Nontuberculous focal infection is of equal importance as a cause of uveitis. Cooperation between internist, oculist and specialists in other fields is necessary. From the Division of Medicine, The Mayo Clinic, Rochester, Minnesota. Read before the Chicago Ophthalmological Society, November 17, 1930.

When a specialist in one department of medicine submits a patient with a lesion or a group of lesions to the internist, he does so because he recognizes that the diseased organ is only a part of the body and is subject to the same disease process as other organs. In fact, he recognizes that the lesion he describes is frequently not indigenous to the special organ but secondary to similar lesions elsewhere and dependent on circulating mediums for existence. His desire then usually is to relate the disease in the special organ to that of the body as a whole, to establish the etiology and to direct treatment intelligently and make recovery possible.

The internist must know something of the criteria required by the specialist in making a diagnosis. He must be familiar with the characteristics of the lesion and he must be conscious of the consistency and inconsistency of appearance and behavior that creates certainty or uncertainty regarding the etiology of the disease. In studying uveitis, he is dealing with lesions of variable character, both as to situation within the pigmentary layer of the eye and as to evolution during the life cycle of the disease in the eye and in the course of the contemporaneous changes within other organs.

These lesions have been recognized and well described for more than two centuries. Etiologic studies have been made, especially in the last century. Virchow described tubercles in the choroid in children who had died of miliary tuberculosis. Manz proved that the lesion in the eye corresponded with similar lesions in the lung, and in 1884, Haab first demonstrated the presence of organisms of tuberculosis within the eye. In 1873, Perls described the first recorded case of tuberculous iridocyclitis of the iris, with miliary tubercles of the retina, and in the next five years Haab described granuloma iridis that was of tuberculous etiology.

As a result of these observations and discoveries a school of workers developed the general thesis that tuberculosis was the primary etiologic factor in many inflammatory diseases of the uvea. They considered that the term uvea included the pigmentary layers of the eye.

In succeeding years, however, organisms of tuberculosis have only rarely been isolated from these chronic ocular lesions and this has caused many ophthalmologists to doubt that tuberculous uveitis exists. Suker and Cushman, objecting to this general denial, remarked: "But the pathological picture is typical of a tubercle in every other respect and the tubercle bacillus has so often been found in and isolated from the tubercle that the identity of tubercle in the eye should not be questioned."

In Lloyd's recent article, he strongly defended the opinion that tuberculosis is the etiologic factor in most of the chronic inflammations which affect the pigmented layer of the eye. He wrote, "The disinclination of the profession to accept tuberculosis as a frequent causative factor and its preference for focal infections as the chief factor

may be explained by the reasonableness of the method of spread in the supposed case of focal disease and the mistaken assumption that lung tuberculosis is similar to the same disease in the eye." This he explained by saying, "The ordinary picture of pulmonary tuberculosis is a late stage of a disease beginning as a pure tuberculous infection of the lungs or bronchial lymph glands, and which does not attract attention as a rule until a secondary infection has caused suppuration and other destructive features that give the clinical signs by which we recognize disease. Rarely inside the eye do we have secondary infection by streptococci and staphylococci."

The internist may not accept as proved a portion of the quotation, especially that dealing with the dependence on secondary infection in the production of symptoms and signs in tuberculosis of the lungs, but he has squarely before him the problem of uveal disease. He cannot avoid making the problem a personal one, and he cannot hold himself apart from the specialist and refrain from lending what assistance he may in solving the question of etiology. It is only by coöperative study and sympathetic understanding that such questions are finally settled. The relation of lesions of the eye to diabetes and to hypertensive disease has been practically settled by specialists, internists and pathologists working together, trusting in one another's sincerity and in the accuracy of their observations.

The specialist must know how the internist thinks and what criteria he demands before he arrives at a conclusion. These requisites can be stated briefly. The internist believes that disease of the uvea is a real entity. He thinks that it may or may not be tuberculous, and that if it is tuberculous, it is almost certainly secondary to tuberculosis elsewhere. He thinks that he has at least a reasonable chance of discovering elsewhere a lesion which in turn will have at least a chance of acting as a primary focus from which, through circulating fluids, bacteria may reach the eye. He will entertain the opinion that perhaps the lesion in the eye may be an allergic manifestation of the lesion he discovers; he will probably reserve judgment in this regard.

The criteria demanded by the internist for diagnosis of tuberculosis

In searching for a tuberculous focus within the lung, there are well-defined criteria to guide the examiner. Heise's criteria apply to two types of patients: those who have a tuberculous lesion but are not made ill by it, and those who have a tuberculous lesion and are made ill by it. The former type he classified as nonclinical tuberculosis, and the latter, as clinical tuberculosis.

To justify a diagnosis of nonclinical tuberculosis, the following criteria must be met: (1) parenchymatous roentgenographically demonstrable lesion must be present and not explainable by other causes than tuberculosis; (2) tubercle bacilli must not be found in the sputum; (3) the upper part of the thorax must be free from persistent râles; (4) the complement fixation test for tuberculosis may be positive or negative; (5) there may be a positive or a negative reaction to the subcutaneous tuberculin test after injection of 10 mg. (once repeated) of old tuberculin and all focal reaction as shown by physical signs (râles) or roentgen ray may be absent; (6) there must be no hemoptysis; (7) there must be no history of unexplained pleurisy with effusion and (8) constitutional symptoms must not be present.

To justify a diagnosis of clinical tuberculosis, one or more of the following must be present: (1) persistent râles in the upper half of the thorax; (2) a parenchymatous roentgenographically demonstrable lesion; (3) bacilli of tuberculosis in the sputum; (4) focal reaction as determined by roentgenologic methods after injection of tuberculin; (5) an unexplained history of hemoptysis, with production of 5 c.c. or more of blood, and (6) an unexplained history of pleurisy with effusion.

These criteria do not include evidence of primary infection, the well-

known Ghon tubercule which may be seen on the roentgenographic film as a calcified peripheral or subpleural lesion. Such infection is associated with calcareous lymph nodes at the hilum.

The criteria do not necessarily include lesions that might escape detection after all possible care had been taken in the search for tuberculosis anywhere in the body but which might cause a positive Mantoux tuberculin reaction. These lesions, on discovery at necropsy, undoubtedly help to make it possible to say that 90 per cent of human beings have had tuberculous infection. It is presumable that not a year passes from birth to death in which human beings do not take organisms of tuberculosis into their bodies.

The criteria do not include those extrapulmonary lesions which occasionally are found in any organ of the body and in the absence of a demonstrable pulmonary lesion. The criteria for their diagnosis is the histologic discovery of a tubercle or the demonstration of organisms of tuberculosis.

The consequence of making diagnoses without criteria or with insufficient standards

All clinicians are now convinced of the fallacy of making a diagnosis of tuberculosis from symptoms alone, in the hope that an early diagnosis might be helpful to the patient. I have expressed the opinion many times that an accurate diagnosis is more desirable than an early one which is based on symptoms alone. A diagnosis should be the result of logical deduction and interpretation of both symptoms and signs of a lesion proved by more exact methods than evaluation of those subtle changes in sound heard or believed to be heard on general examination.

Specialists may deplore, as I do, a decline in the skill of physicians in making examinations of the thorax, but one should recall that the roentgenographic plate, which first demonstrated a pulmonary lesion, did more to enforce accuracy and to establish an honest knowledge of the limitations of general examination than had been accomplished in centuries.

The records of the armies of various nations show that, without the use of accurate diagnostic measures, accuracy of diagnosis occurred in only 18 to 22 per cent of all men with arrested, cured, or active tuberculosis who were reëxamined. Clinicians have been led to believe that the presence or absence of râles in the upper part of the thorax might be accepted as evidence for or against a diagnosis of tuberculosis. If one views the matter from the standpoint of Heise, then in mild cases, with positive roentgenologic changes, the absence of râles would have brought about error in 41 per cent of cases, in moderately advanced cases in 25 per cent, and in far advanced cases in 11 per cent. These groups of proved cases named, failed to have this well-known sign, in which confidence is most frequently placed in general examination.

The internist, therefore, presents the criteria for diagnosis in the hope that the specialist may know how he thinks and what exacting demands he makes before he reports on the presence or absence of tuberculosis in any case of ocular disease submitted to him for study.

The internist's view regarding toxic or allergic phenomena

Krause, in the course of experimental studies in allergy, made a general statement that is acceptable, understandable and explanatory of phenomena that may develop in a person who has been sensitized by a first infection which did not produce symptoms itself. "For our purpose, immunity is regarded as that condition of increased specific resistance to implantation and extension of tubercle bacilli, and allergy as that state of tissue hypersensitiveness to tuberculo-protein that comes into existence with the earliest formation of the specific anatomical tubercle. Both immunity and allergy continue to work within the body as long as the tubercle and its tubercle bacilli persist."

Suker and Cushman made use of this acceptable hypothesis and wrote:

"This allergic reaction to tubercle bacilli seems to provide a suitable theory for many ocular manifestations of tuberculosis and indeed, seems to explain why satisfactory results are obtained in the use of tuberculin in cases of a chronic tuberculous nature, while it is of no value in the acute miliary form."

The clinician cannot find objection to this theory because it presupposes the existence of first infection, of reaction of tissue to secondary infection and therefore of a lesion which can almost always be discovered. Without a lesion, the clinician must maintain a skeptical attitude and hold that there is insufficient evidence to warrant a positive diagnosis of tuberculosis. It may be argued that similar lesions of nonspecific character, with absence of the structure and components of tubercle may be found in the skin. These tuberculids are known under many names: erythema nodosum, erythema induratum, and papule-necrotic tuberculid of which folliclis and acnitis are varieties.

It may still be open to question whether the skin tests for tuberculosis are really specific and cause an allergic reaction in the skin of inflammatory character due to sensitivity developed in tuberculous first infection. It may still be possible that the effects of injections of tuberculin used in treatment may be due to the action of foreign protein and not specifically to a response of a sensitized tissue to tuberculo-protein. At present, it would seem that the clinician cannot assume a dogmatic attitude regarding these controversial questions. His duty is to report the presence or absence of a lesion that satisfies the criteria he demands for diagnosis. Then he clears the situation to the satisfaction of himself, and usually to that of the specialist. He must not be afraid to say he does not know or to report that he cannot make a positive diagnosis when the specialist sends him a patient who has a lesion that seems to represent either a tuberculous deposit in the eye or an allergic result in the eye of tuberculosis elsewhere.

My attention at this time is chiefly directed to the study of tuberculosis as the etiologic factor in ocular diseases affecting the uvea. It should be understood that investigation of nontuberculous focal infection within the body is of equal significance. It becomes the anxious concern of the examiner in every instance. The standards of examination are quite as exacting. The clinician himself inquires into the past and present history of disease, and he satisfies himself regarding the status of all the structures concerned. He is required to know sufficiently well the technique of special examinations to permit him to distinguish normal from abnormal characteristics. He employs the roentgen ray to disclose hidden disease in the accessory sinuses in the head, in the gallbladder, and in the intestines, even though he may in his own mind question the possibility of metastatic or embolic spread to the eye of disease from the region examined. He has palpable proof of the condition of the prostate gland in men and of the cervix in women and he employs microscopic and bacteriologic study to determine the character of the discharges from them. He calls to his assistance the skill of specialists in each particular field and advises inoculation of animals in the hope of establishing a connection between the organ studied and ocular disease by reproducing a similar lesion in the animal. He uses autogenous vaccines in treatment in the hope of producing either a specific or a foreign protein reaction and a resulting improvement in the ocular lesion. He does not hesitate to remove organs such as teeth, tonsils, gallbladder, or cervix when the ocular lesion is severe and there is sufficient evidence to warrant the belief that one bears an etiologic relationship to the other. Nor is he satisfied with measures of this kind only. He advises the specialist regarding the daily life and hygienic management of the patient, prescribing a regimen not unlike that employed in treating other chronic diseases. The internist is not assured that recrudescence may not appear and he feels certain that the element of physical fitness is

important. He looks on ocular disease as a condition similar to other secondary diseases within the body and he is not unwilling to believe that an ocular lesion, long after all external foci have been extirpated, may become reactivated if general health becomes impaired. The clinician is not unwilling to think that the lesion passes through various metamorphic changes until its investment becomes complete, symptoms are no longer present, and the disease is either quiescent or cured. He is not averse to thinking that such a lesion, during such metamorphic changes, may bear within itself the elements for its own reactivation.

The pathologist's opinion acceptable to the clinician

Throughout the investigation, the internist is interested chiefly in pathologic manifestations of disease; he looks for a lesion that may be demonstrable; without that the diagnosis is clouded. He becomes interested also in the pathologist's opinion regarding lesions of tuberculosis in general and of the eye in particular. One well-known neuropathologist of wide experience reported to me that he had examined, following operation, only one eye in which he was able to demonstrate a tubercle. He insisted that tuberculosis in the eye is a rare pathologic entity and is almost certainly a secondary disease. He was equally insistent that it is the same as tuberculosis in any other organ, that it produces a typical lesion made up of giant cells, epithelial cells and round cells, that it contains organisms of tuberculosis, that it demonstrates the changes of necrosis and fibrosis, denoting the variable factors of time and of resolution. The pathologist examining a fibrous lesion of the eye would not feel justified in making a diagnosis of tuberculosis unless most of these conditions were seen even though tuberculosis were present in the lung. He would make a diagnosis of chronic inflammatory disease of undifferentiated etiology. He would not feel safe in making a diagnosis of tuberculosis of alergic or toxic type.

The pathologist might concede the possibility of such a lesion when tuberculosis could be found elsewhere, if these lesions might reasonably be expected to be activated enough to cause allergic or toxic reaction. He would wonder, however, why such cases are not found in sanitariums where large numbers of patients with tuberculosis are grouped, are most systematically studied and are under the closest observation.

Comment

It seems unfortunate that clinicians are not more greatly interested in diseases of the eye and better trained in methods of examination of the eye and the use of the ophthalmoscope. It is equally unfortunate that neuropathologists have such limited experience in the examination of eyes. With more nearly routine examination, they might recognize not only the solitary tubercle associated with miliary tuberculosis but examine other lesions by the usual methods; they might employ inoculation of animals as a test, and, above all, they might tabulate and carefully describe the lesions they see. In the future, when coöperative effort in the diagnosis of these uveal inflammations permits the work of clinicians and men in special fields to be compared, correlation will be possible and diagnosis will be acceptable to all because it will be based on as firm a foundation as that which makes retinitis of pernicious anemia and leukemia a diagnosis acceptable without question.

The internist would welcome cooperative work. He would like to know the criteria of diagnosis acceptable to the specialist. He would like to know how the specialist approaches his problem, and what he thinks about it, and he would be happy to discuss his observations and what he knows about systemic disease with frankness. Just now, it is possible to recognize as tuberculosis a few lesions of the uvea. In the future, there may be others equally recognizable. At present, accurate description is better than names, and observation and experimentation, better than custom and authority.

The Mayo Clinic, Rochester, Minn.

THE PATHOLOGY OF PAPILLEDEMA

An examination of forty eyes with special reference to compression of the central vein of the retina

W. E. Fry, M.D., D.Sc. (Med)*
PHILADELPHIA

An intensive study was made of forty human optic nerves cut in serial sections. The cases included brain tumor, meningitis, brain abscess, gumma of the brain and aneurysm. Thirty-two of the eyes exhibited papilledema. The specimens had been carefully removed at autopsy and in twenty-six instances the posterior part of the eye with the optic disc cut in longitudinal section was studied. In the majority of cases of papilledema there was compression of the vein along the side of the nerve or in the subarachnoid space. It was concluded that venous stasis thus produced is the initial cause of papilledema. There was evidence that forward pressure of fluid within the optic nerve from infiltration of cerebrospinal fluid is a secondary cause. No evidence was found of an inflammatory or toxic cause. From the Department of Ophthalmology of the University of Pennsylvania.

The striking ophthalmoscopic appearance that is characteristic of papilledema has been of great clinical interest since the description of retinal hemorrhages in a case of brain tumor by Türk[1], in 1853. It was not until seven years later, in 1860, however, that Graefe[2] first described papilledema and pointed out its relationship to intracranial disease. The English terminology for this condition has been somewhat confusing. The term choked disc is Clifford Allbutt's translation of "Stauungspapilla". The use of such terms as papillitis and optic neuritis has led to the confusion of this condition with those in which there is some haziness of margins of the optic disc, with perhaps some swelling of the disc itself, but which have an entirely different etiology and significance. We owe to Parsons the introduction of the term papilledema.

There have been numerous clinical statistics and individual case reports in regard to papilledema, but the contributions which deal with the pathologic examination of a series of cases are limited in number. Such examinations have been made by Elschnig[3], twenty-six eyes; Kampherstein[4], fifty-one eyes; Paton and Holmes[5], sixty eyes; Schieck[6], six eyes; Wilbrand and Saenger[7], fifty-four eyes; and Behr[8], forty-five eyes.

The theories in regard to the pathogenesis of papilledema may be divided into the mechanical and the nonmechanical. They may be further subdivided. The mechanical types may have to do either with a disturbance of the lymph flow or with an interruption of the vascular supply of the optic nerve and papilla. The nonmechanical types may be either inflammatory or toxic. The first theory advanced was by Graefe, in 1860, who believed that the brain tumor either directly or indirectly pressed upon the cavernous sinus, and caused edema of the nerve, venous engorgement, and retinal hemorrhages because of the venous stasis produced in the ophthalmic vein. An inflammatory theory was formulated by Gowers[9], as early as 1879, and several years later, in 1881, Leber[10] stated the cause to be the development of a toxic material by the intracranial disease. A direct forcing forward of an edema originally present in the brain was the cause advanced by Parinaud[11], Ulrich[12], Sourdille[13] and Kampherstein.

In 1869, Schwalbe[14] demonstrated the connection between the subarachnoid space of the brain and that of the optic nerve. This fact was the starting point of many theories of the mechanical category. Two of the most important of these were by Schmidt-Rimpler[15] and Manz[16]. The former believed that there was a direct driving forward of the cerebrospinal fluid through the lamina cribrosa, with an edematous in-

* Thesis submitted to the Faculty of Ophthalmology of the Graduate School of Medicine of the University of Pennsylvania in partial fulfillment of the requirements for the degree of Doctor of Medical Science (D.Sc. (Med.)) for graduate work in Ophthalmology.

filtration of the nerve head as a direct result, while the explanation of the latter was based upon compression of the central vessels of the retina as they passed through the lamina cribrosa. Cushing and Bordley[17] have produced experimental work that supported the views of these two authors and state their position as follows, "we believe that the mechanical views of Schmidt-Rimpler and Manz are largely correct and that the lesion is primarily due to the crowding into and distension of the sheath of Schwalbe by the obstructed cerebrospinal fluid, resulting in an edema of the nerve head, in which toxic elements play an insignificant part." A different type of explanation was advanced by Schieck[18] who believed the condition was caused by the entrance of the cerebrospinal fluid under increased pressure in the preformed perivascular lymph spaces of the axial bundle of the optic nerve and along the central vessels into the disc.

Compression of the central vein of the retina has been assigned a primary importance in the production of the condition under discussion by Deyl[19], Dupuy-Dutemps[20], Merz[21], Knape[22] and Paton and Holmes[5]. These authors disagree, however, about the place at which this compression takes place Deyl and Knape believed the compression took place where the vein passed through the dura, while Dupuy-Dutemps, Merz, and Paton and Holmes believed the vein was compressed in the intervaginal space.

The present observations are based upon the examination of a series of forty eyes obtained from twenty-one cases. These cases were from the services of Dr. C. H. Frazier, Dr. William Spiller, Dr. Alfred Stengel and Dr. Gittings, at the hospital of the University of Pennsylvania. Six of the cases were in females and fifteen in males. The oldest patient was fifty-three years of age and the youngest was four years. The average age was twenty-nine years. In seventeen of the cases the diagnosis was brain tumor, in two of the cases meningitis, and there was one case each of brain abscess, gumma of the brain and one aneurism. In thirty-two of the eyes examined there was a papilledema present and in all but two of these the condition was recent. In these two eyes there was a subsiding papilledema, but the swelling still measured three diopters in height. In one of the cases the swelling amounted to nine diopters. The average swelling was three and nine-tenths diopters. Two of the cases showed a unilateral papilledema. In thirty-seven of the cases the optic nerve was cut in transverse serial sections. The sections were cut twenty microns thick. In some instances every fifth and in other instances every tenth section was prepared and mounted for examination. The length of the nerve that was sectioned varied in the different instances. The sectioning in three nerves began at the level of the highest point of swelling of the disc. In the other cases the sections began about 3 mm. behind the globe. In several of the series the sectioning was continued into the intracranial portion of the nerve. An average of about one hundred and fifty sections was examined for each nerve. In twenty-six instances the posterior part of the eye, containing the optic disc was cut in longitudinal section. The majority of the sections were stained with eosin and hematoxylin, but in addition some of the sections were stained with Weigert's and with van Gieson's stain. Care was taken in removing the specimens at the autopsy. In all the cases the fat and muscle surrounding the nerve were allowed to remain undisturbed until after the specimen had been hardened in formalin. In many of the cases the bony structure of the optic foramen was also allowed to remain until the specimen had been hardened.

Particular attention in this series of observations was given to the intraneural course of the central vessels of the retina. This was done because of the clinical importance that has been attached to retinal venous engorgement and retinal hemorrhages, because of the numbers of theories of papilledema, as noted previously, that are based upon a local compression of the central vein of the retina, and because of the varia-

tion in the points at which different authors believe that the compression takes place. The relationship of the increased intracranial pressure to an increased retinal venous pressure has been pointed out by Baurmann[23] and to increased retinal arterial pressure by Conrad Berens[24].

In a previous contribution[25] attention has been directed to certain variations of the intraneural course of the central artery and the central vein of the retina. The observations contained in this contribution were made during the examination of the present series of optic nerves. Because these variations occurred with such regularity and in such a considerable percentage of the cases examined, because they differed markedly from the usually described course of the central vessels, and because of the importance of the relationship between venous congestion and papilledema, it was considered of importance to review these variations in some detail at this point.

According to the observations contained in the contribution referred to above, we may distinguish two types of vessel arrangement. The artery and vein pass directly posteriorly through the center of the nerve, usually close together, and at a variable distance behind the lamina they turn acutely and proceed toward the periphery of the nerve. The two types of vessel arrangement depend upon the course at this point. We may speak of type one in which both the artery and the vein proceed to the same point in the arc of the periphery of the nerve, and of type two in which they proceed to different points on the arc of the nerve. In this second type, the artery and the nerve diverge from each other usually at an angle of ninety degrees, although it may be slightly less or greater than this in some instances. Type one occurred in forty-two per cent of the instances and type two in fifty-eight per cent of the instances.

In the first type, in which the artery and vein proceeded to the periphery of the nerve at the same point, either the artery or the vein might be slightly anterior, and usually both vessels immediately crossed the intervaginal space and pierced the dura. In one instance, however, the vein had a subpial position for a distance of 0.9 mm. and in another instance for 3 mm. In the second type, in which the artery and vein diverged from each other at an angle of ninety degrees, the vein usually approached the periphery of the nerve anteriorly to the artery. It then ran posteriorly within the pial sheath for a variable distance before crossing the intervaginal space. In one instance this distance amounted to 8 mm. The artery usually crossed the intervaginal space immediately upon appearing on the periphery of the nerve, and in all instances crossed the space before the vein, even though the latter had appeared in a subpial position while the artery was still within the center of the nerve. In four nerves, the vein, after coursing posteriorly in the pia, turned at right angles about the periphery of the nerve, and passed through an arc of a quarter of a circle to make its exit from the dura at a point corresponding to the line of the exit of the artery.

As far as it has been possible to determine, these facts in regard to the anatomical relations of the central vessels of the retina had not been previously pointed out. The relatively long subpial and intravaginal position of the central vein is of importance in any condition of an increased pressure of the fluid in the subarachnoid space of the nerve, rendering the vein particularly liable to compression at this point.

The evidence in regard to venous compression has been studied in detail, and the importance of this has been previously noted. For this purpose thirty-seven nerves were available and of these thirty-one were from eyes showing papilledema and six were from eyes that did not show papilledema. The central vein is easily followed from a position just anterior to the lamina cribrosa. The lumen is well dilated at this point and shows no constriction at the lamina cribrosa. It continues to be well dilated in its course posteriorly through the central part of the nerve stem. In all but four cases

the lumen was dilated while the vein was within the nerve stem, and even in these cases it was not reduced to a slit-like character. In addition, in spite of the care taken not to traumatize the tissue at the time of the autopsy, there were other changes in the nerve which indicated that this could have been produced by an artefact; namely, a flattening of the nerve. At a variable distance behind the lamina the vein makes an acute turn and approaches the periphery. When the vein first appears on the periphery of the nerve, the lumen is widely dilated.

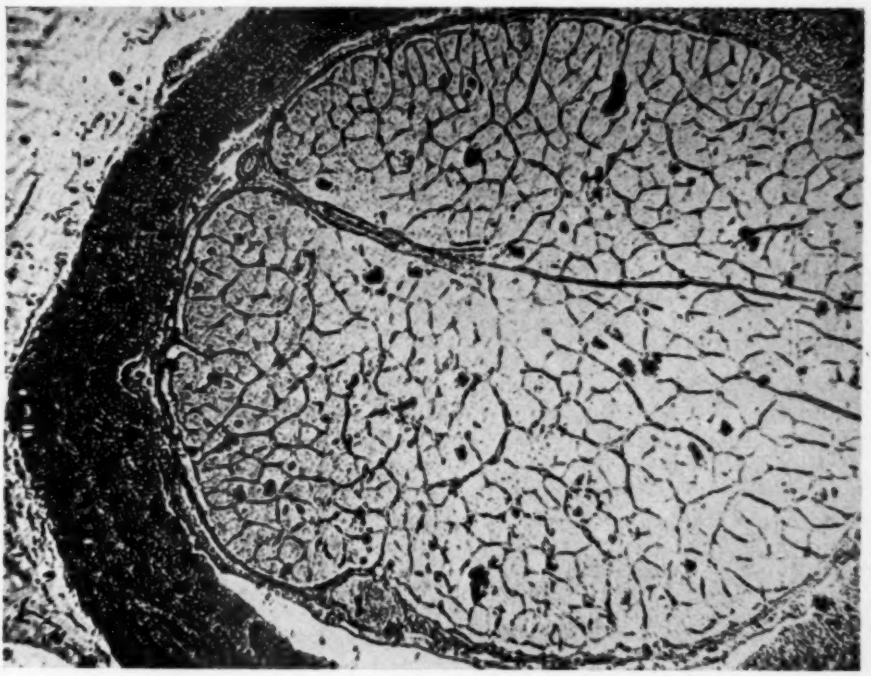


Fig. 1 (Fry). Transverse section of the nerve stem, showing the vein below on the periphery of the nerve and well dilated. The artery runs horizontally to the left and comes to the periphery of the nerve at a point ninety degrees from the point at which the vein came to the periphery.

The vein, in the cases that fell under type two continues downward along the edge of the nerve for a variable distance. In several instances this amounted to as much as 8 mm. The vein, in the instances that fell under type one, proceeds to cross the intervaginal space almost immediately upon reaching the periphery of the nerve. Evidence of marked compression of the lumen of the vein is found either on the side of the nerve or within the subarachnoid space. In the majority of the cases of papilledema the lumen was entirely obliterated; only a double line of endothelial cells marking the position of the vein. At these points the position of the vein is noted with difficulty, and in many of the preparations it would have been completely overlooked, had not serial sections been followed. The distribution of the nerves in regard to the amount of venous compression is indicated in table one. Of the thirty-one nerves from eyes show-

Table I.

DISTRIBUTION IN REGARD TO THE AMOUNT OF VENOUS COMPRESSION

	No. of cases	Showing slit-like compression	%	Non slit-like compression	%
Showing papilledema	31	26	84	5	16
Not showing papilledema	6	3	50	3	50

ing papilledema, twenty-six, or eighty-four per cent showed marked compression with the lumen of the vein reduced to a slit, while five, or sixteen per cent showed evidence of compression but not to such a marked degree. In the six cases not showing papilledema, there were three cases showing compression of the vein, and three cases not showing compression of the edge of the nerve while the vein was in the pia, or in the subarachnoid space. The distribution of the nerves in this regard is given in Table II. In the twenty-six nerves that showed marked compression and papilledema, the compression took place while the vein was on the margin of the nerve in ten instances, or thirty-eight per cent, and it took place in the subarachnoid space

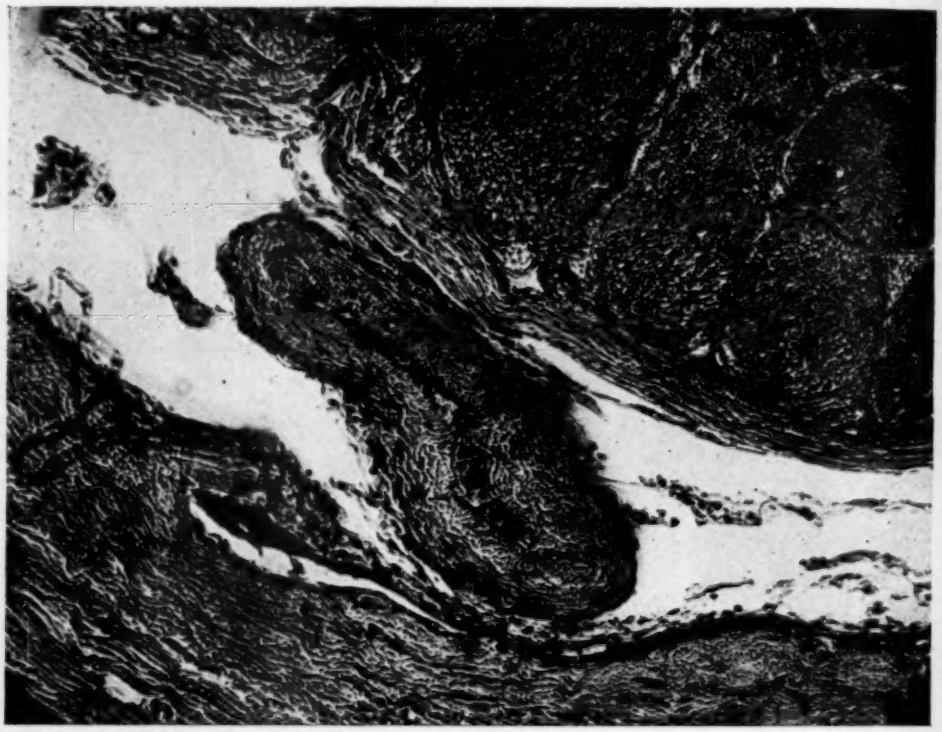


Fig. 2 (Fry). Marked compression of the central vein of the retina when it is in the subarachnoid space.

vein, namely, fifty per cent in each instance. This indicates that there must be some local external pressure, the increased cerebrospinal fluid in the subarachnoid space of the nerve, that produced the venous compression in the nerves with papilledema.

The point at which the marked compression took place was either along the in sixteen instances, or sixty-two per cent. In the three cases not showing papilledema, but showing some compression, this compression was while the vein was in the pia in two instances, and in the subarachnoid space in one instance. Here again the evidence is in favor of an active compression of the central vein by an increased fluid pres-

Table II

DISTRIBUTION IN REGARD TO THE POINT AT WHICH SLIT-LIKE COMPRESSION TAKES PLACE

	No. of nerves	On the side of the nerve	%	In the subarachnoid space	%
Showing papilledema	26	10	38	16	62
Not showing papilledema	3	2	67	1	33

sure, and not due to an artefact in the preparation or to an anatomical type.

The marked compression of the central vein may occur either when the arrangement of the central vessels is as in type one or type two of the above classification. Of the twenty-six instances of nerves with papilledema and marked compression, ten, or thirty-eight per cent fell under type one, and sixteen, or sixty-two per cent fell under type two. There were five nerves showing papilledema in which marked compression was not noted. Four of these, or eighty per cent, fell under type one, and one, or twenty per cent, fell under type two. This would indicate that the type of the vessel arrangement has a direct bearing on the amount of venous compression that is exerted, and may help explain why, in cases of papilledema, with equally increased intracranial pressure, one may show many retinal hemorrhages while the other may show few or none.

The edema of the nerve head is the finding which chiefly accounts for the ophthalmoscopic and gross appearance of the papilla, that is characteristic of papilledema. This edema is most marked anterior to the anterior portion of the lamina cribrosa and becomes progressively more marked anteriorly and laterally. It is the edema of the more laterally placed fibers of the disc that causes the *s*-shaped course of the fibers here and causes the displacement of the retinal layers. The edema continues only for a short distance into the fiber layer of the retina. In but one disc was any notable amount of cellular infiltration present. This was from a case of gumma of the brain.

Fig. 3 (Fry). Periphery of the optic nerve showing the distended central vein of the retina when it first appears in a subpial position.

There has been much discussion in regard to the site and amount of edema of the nerve stem. In the present series of cases the edema of the nerve was found to be subpial, about the central vessels, and interfascicular in location. The perivascular and subpial types

were the most frequently seen. It is very difficult to judge the presence of a small amount of edema or to determine, as some authors state, that the amount of edema rapidly diminishes behind the point of exit of the central vessels. With the view of determining this latter point twenty-four nerves showing papilledema were measured. The average diameter was obtained at a point between the globe and the point of exit of the central vessels, and again at a point between the exit of the central vessels and optic canal. In six of these nerves the diameter anterior and posterior to the position of the exit of the central vessels was the same, and in one instance the diameter posterior was greater. In the remaining seventeen instances the anterior diameter was greater. The average diameter of the twenty-four nerves anterior to the place of exit of the vessels was 3.41 mm. and the average diameter posterior to this place was 3.25 mm. This gave an average difference of .16 mm. This would indicate that there was a somewhat greater amount of edema of the nerve stem anteriorly.

The sheath of the optic nerve was distended in twenty-four of thirty-one cases, seventy-seven per cent. In nineteen of the nerves, sixty-one per cent, the distention was marked. The subarachnoid space was the one involved. Usually the arachnoid was closely applied to the dura. In some of the nerves, subarachnoid hemorrhage was present, and in several instances both subarachnoid and subdural hemorrhage were present. The hemorrhage in these cases was post operative. In the cases in which subarachnoid hemorrhage was present the red cells could not be traced into the nerve stem with the central vessels or about the disc from the anterior extremity of the subarachnoid space. This latter finding corresponds to the findings of Friedenwald who examined several such cases in serial section and was not able to demonstrate a connection between the retinal and the subarachnoid hemorrhages. In two instances there was a purulent exudate in the subarachnoid space. These were from a case that had developed a secondary meningitis. A varying amount of endothelial proliferation was seen, but in only two instances was this excessive in character.

A cross section of the disc shows the characteristically shaped swelling of the papilla. In addition to the edema that has been previously described, there are three notable features. These are the marked forward arching of the anterior portion of the lamina cribrosa, the frequent preservation of the physiological cup, and evidence of pressure from behind forward at the entrance of the optic nerve into the globe. It was possible to examine eighteen of the nerves with papilledema in regard to the position of the anterior fibers of the lamina cribosa, and in seventeen, or ninety-four per cent of these, it was arched forward. In none of the cases not showing papilledema was this found to be true. This arching of the lamina cribrosa fibers divides the cross section of the disc into two parts that differ in the extent of edema exhibited, namely, the part posterior and the part anterior to the arching fibers. The latter portion although less in extent, usually shows the more marked amount of edema. The former portion is greater in extent, and is the portion which chiefly accounts for the mushroom like shape of the papilla. The appearance given is not that the lamina cribrosa fibers alone have been forced forward, but that the section of the nerve between the two portions of the lamina has been elongated. The second feature noted above contributes to this view. The evidence of a forward pressure at the entrance of the optic nerve into the globe is shown in two findings. The sections of well marked papilledema show a turning forward of the inner layers of the sclera, together with the lamina vitrea and the pigment layer of the retina. In one nerve the pressure was so great that the lamina vitrea had been turned inward and folded upon itself. These two features indicate that the forward acting pressure was much greater than could be exerted by a simple retardation of the normal lymph flow from within the globe into the nerve and its perivascular lymph spaces.

In the present series the degenerative changes have not been studied. Such changes have been recorded in detail by Paton and Holmes and by Behr. Although these changes help to explain some of the clinical findings, such as concentric contraction of the visual fields, they do not aid particularly in determining the method of production of papilledema.

anterior to the arched fibers of the lamina cribrosa. This also causes the overfilling and tortuosity of the veins seen on ophthalmoscopic examination. This feature along with the blurring of the disc margins which forms the initial stages of a papilledema indicates that this venous compression is the first factor in point of time. The second factor is a forward pressure at the optic en-

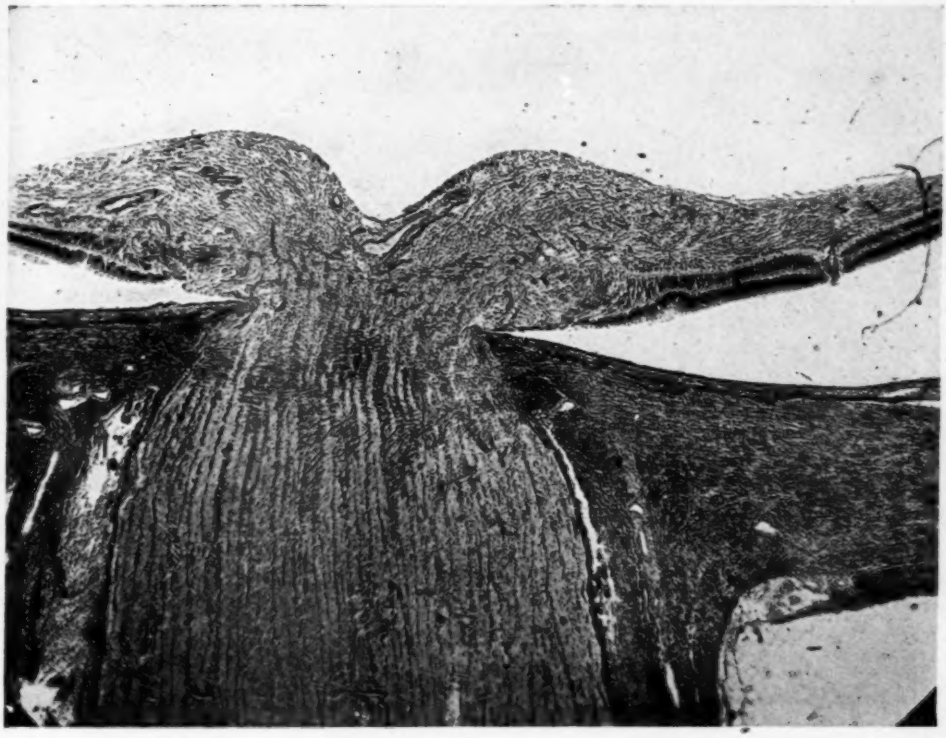


Fig. 4 (Fry). Longitudinal section through an optic disc showing papilledema. It shows the displaced retinal layers, the preservation of the physiological cup, the arching of the anterior lamina cribrosa fibers and distended vessels.

The above description indicates the lack of an inflammatory or toxic cause of papilledema and favors the operation of a mechanical cause. From these examinations it may be stated that there are two factors operative in the production of papilledema; first, a compression of the central vein of the retina either along the periphery of the nerve or within the subarachnoid space by the increased fluid pressure of the space. This causes an increased transudation of lymph from the vessels and probably accounts for the more marked edema

trance into the globe from within the nerve stem and sheath. The present examinations do not indicate the mechanism by which this forward pressure takes place, although, in all probability, it is due to the increased cerebrospinal fluid pressure in the nerve sheath with a direct infiltration of the papilla.

A comparison of the finding listed above with the various theories of papilledema that have been previously outlined shows that there is no evidence of the presence of an inflammatory or toxic agent as the immediate cause. The

slight inflammatory change that may be present, is, in all probability, secondary to degenerative changes. In the three instances in which inflammatory changes were evident, there were other factors present, namely, syphilis in one case and a secondary meningitis in the others. There is also no evidence for a general vasomotor etiology. On the other hand, there is definite evidence

tors. There is ample reason, in the slit-like compression of the central vein, the distended nerve sheath and the forcing forward and even turning over of the lamina vitrea at the margin of the disc, to believe that the cause of papilledema is not a compression of the nerve at the optic foramen as held by Behr.

In regard to the second factor that is operative in the production of papil-

Fig. 5 (Fry). Longitudinal section through a portion of a disc showing papilledema showing the lamina vitrea curled backward upon itself and the well marked arching of the anterior fibers of the lamina cribrosa.

of a local obstruction to the circulation of the central vein of the retina. In this respect the findings correspond to those of Paton and Holmes, Dupuy-Dutemps, Deyl and Merz who placed the point of obstruction on the side of the nerve and in the subarachnoid space. The findings do not agree with those of Knape, who placed the point of obstruction in the dura, or with Manz who believed that an obstruction took place at the level of the lamina cribrosa. There is also an agreement with Paton and Holmes who believe that the venous obstruction is the initial of two factors.

ledema, the forward bowing of the anterior portion of the lamina cribrosa, with the appearance not only of edematous infiltration but of elongation of the portion of the papilla between the two different parts of the lamina cribrosa, indicates that there is some factor other than a blocking of the normal lymphatic drainage from the vitreous chamber into the lymphatic system of the optic nerve as held by Paton and Holmes and by Berens. Although the present examinations do not show a direct connection of the subarachnoid space with a lymphatic system of the

papilla, the opinion is advanced that there is a direct forcing forward of the cerebrospinal fluid of the intervaginal space under increased pressure and that the entrance is probably by means of the perivascular lymphatic spaces. This corresponds to the view held by Schieck.

In summary the following statements may be made.

1. No evidence was found for an inflammatory or toxic cause of papilledema.

2. The initial cause of papilledema is a compression of the central vein of the retina either along the side of the nerve or in the subarachnoid space. This produces a venous stasis with a consequent transudation of lymph into the tissues of the optic disc and adjacent nerve fiber layer of the retina.

3. The secondary cause is a forward pressure of fluid within the optic nerve, produced in all probability by the infiltration of cerebrospinal fluid under increased pressure in the perivascular lymphatic spaces.

1912 Spruce Street.

References

[1] Türk. Cited by Parsons.
[2] Graefe. Arch. f. Ophth., 1860, v. 7, pp. 58-71.
[3] Elschnig. Arch. f. Ophth., 1895, v. 41-2, pp. 179-293.
[4] Kampherstein, Klin. M. f. Augenh., 1904, v. 42, pp. 501-525.
[5] Paton and Holmes, Brain, 1911, v. 30, p. 389.
[6] Schieck. Die Genese der Stauungspapille, Wiesbaden, 1910.
[7] Wilbrand and Saenger. Neurologie des Auges, Wiesbaden, 1909, v. 4, p. 751.
[8] Behr, C. Arch. f. Ophth., 1920, v. 101, pp. 165-209.
[9] Gowers. Medical Ophthalmoscopy, Phila., 1904, pp. 74-87.
[10] Leber. Trans. First Internat. Cong. London, 1881, v. 3, pp. 53-57.
[11] Parinaud. Ann. d' Ocul., 1879, v. 82, pp. 1-46.
[12] Ulrich. Arch. f. Augenh., 1887, v. 17, pp. 30-54.
[13] Sourdille. Ann. d'Ocul., 1901, v. 21, pp. 378-399.
[14] Schwalbe, Arch. michr. Anat., 1870, v. 6, pp. 1-61.
[15] Schmidt-Rimpler. Arch. f. Augenh., 1888, v. 18, pp. 152-170.
[16] Manz, Deutsch. Arch. f. klin. Med., 1872, v. 9, p. 339; Manz, Monatsbl. f. Augenh., 1865, pp. 281-284.
[17] Cushing and Bordley. J.A.M.A., 1909, v. 52, p. 353.
[18] Schieck. Arch. f. Ophth., 1924, v. 113, pp. 157-159; Schieck. Deutsche Med. Woch., 1913, p. 10.
[19] Deyl. Wiener klin. Rundschau., 1899, v. 13, pp. 165-167.
[20] Dupuy-Dutemps. Paris Thesis, 1900; Dupuy-Dutemps. Bul. Soc. d'Ophth. Paris, 1929.
[21] Merz. Arch. f. Augenh., 1900, v. 41, pp. 325-355.
[22] Knape, E. Mitt. a. d. Augen. d. Carolinsch Medico Chru. Institut zu Stockholm. 1909, v. 10, pp. 1-24.
[23] Baurmann, Deutsche Ophth. Gesell., 1912, v. 36, pp. 14-22.
[24] Berens, Smith, and Cornwall. Arch. Neurol. and Psychiat., 1928, v. 20, pp. 1151-1171.
[25] Fry. Arch. of Ophth., 1930, v. 4, pp. 180-187.

XERODERMA PIGMENTOSUM

F. B. Blackmar, M.D.
COLUMBUS, GEORGIA

This rare disease is described and one case added to the literature.

Xeroderma pigmentosum is a fatal skin disease which begins in infancy, usually in the first year. Although it is classed as a malignant disease and results in death after ten or fifteen years, it rarely metastasizes to internal organs.

The usual history is that sometime in the infant's first spring or summer after more exposure to sunlight than usual (but still no more than the average child could easily tolerate) he was badly sun burned. After this freckles appeared on the face, hands, and less on the legs. In other words wherever the skin was not protected by clothing. During the first or second winter the child may seem to be entirely recovered only to have a flare-up in the summer. The effect of strong light is especially painful to the eyes. As the child grows older he hides his eyes when exposed to light. Warty growths appear after a few years on some of the pigment spots and grow to large size unless curetted off deeply. Such treatment is usually successful and safe although of course scars are left at the site of the removal. Angiomata are said to develop but neither of two cases seen by me gave any evidence of these. The favorite site for these growths is at the mucocutaneous junctures as, the lid margins, the end of the nose and the mouth. Much annoyance follows contractions of scars left by the removal of these growths. The opening of the nose may thus become obstructed. While the epitheliomata are growing on the lids these are turned in against the cornea. Later the lids are partially destroyed and partially dragged away from the eye ball leaving the cornea unprotected. Ulcerative keratitis develops and is followed by opacities of the cornea and more or less blindness. After six to ten years the malignant character of the condition begins to manifest itself by malnutrition from the drain on the body resources and death follows.

This disease was described in 1870. In the literature to 1920 only eighty cases had been noted.

Etiology: A claim has been made that one factor is the inter-marriage of relatives. In neither of my two cases were the parents related. The belief that heredity is concerned may be strengthened by the fact that often more than one child in the same family is affected. Because the lesions are limited to the areas of skin which are most exposed to light it is claimed that there is a congenital hypersensitivity to ultraviolet rays. In connection with this angle of the disease it was interesting to me to observe that while the patient, whose case is here reported, was being photographed under a mercury vapor arc light, which was free from any sensation of heat, she was entirely comfortable. However, when an ordinary high power electric light bulb with a reflector was turned on, in trying to get more detail in the picture, it could not be tolerated at all. The feeling of heat on the patient's face from the incandescent bulb was quite noticeable to my hand supporting the patient's head.

Treatment: There is no curative treatment which can be relied upon although one case was reported cured by x-ray. In connection with this it should be borne in mind that these patients are particularly sensitive to soft rays but their tolerance to hard rays may be even greater than normal. Arsenic, mercury, iodides, and cod liver oil have been used without effect. As the growths appear they should be removed with a curette. The sun proofing ointments might prove useful when an exposure to the sun cannot be avoided.

Case report: F. C. a white female aged six years, was brought to me for advice in reference to an ulcerated growth on her right lower eye lid and a skin disease of her face and hands.

Family history: The patient is one of three children. An older brother is

in good health. Another brother died of pneumonia at the age of sixteen months. This brother when three weeks old scaled as though blistered, after any exposure to sun light. Freckles appeared on all exposed parts of the body. He passed through only one summer before contracting a fatal attack of pneumonia. The patient's paternal great grandfather died from a cancer of the eye. There was no history of malignancy in the mother's family. Frequent Wassermann tests have been run on the parents with negative results. Both parents are in good health and are not blood relations. Neither are either freckled now nor were they in their childhood.

Past history: The child's birth was normal at full term. She has been entirely healthy and has not suffered from any childhood diseases. There have been frequent colds in the past but so far she has been free from them this winter.

Present illness: In early spring at the age of six months the mother noticed that the baby appeared to be badly sunburned on one cheek after sitting in her crib on the front porch. Large vesicles soon formed. These spread over the baby's entire face as the summer advanced. There was some burning of her hands and a little effect on her feet. At one time the hands were more affected than the face. A warty growth appeared on her right cheek and two on her neck. One of the growths on her neck was two inches in diameter and one inch high. This was removed by a physician with a high frequency current but recurred after one month. The mother purchased and applied a caustic paste which finished it's destruction.

The present growth on the eye lid appeared three months ago. It has slowly increased until it now occupies almost the entire lid, being elevated about half an inch. The eye ball seems as yet unaffected. Photophobia has been marked

(Blackmar). Xeroderma pigmentosum.

all the patient's life. She cannot tolerate day light enough to join in games and on account of her appearance is shunned by other children.

Examination: The right lower lid is covered by a red ulcerated knotty elevated growth. The patient is a mouth breather due to adenoids. The skin on her face is tight and glistening. There are many scars on the backs of her hands, face and neck. Large freckle-like, pigmented areas are scattered over her face. The skin on her abdomen and chest is normal.

Swift Kyle building.

DEEP ORBITAL INJURY BY STICK OF WOOD: EXOPHTHALMOS AND OPTIC ATROPHY FOLLOWING

H. L. HILGARTNER, M.D.

H. L. HILGARTNER, JR., M.D.
AUSTIN, TEXAS

A large wood fragment which had penetrated the orbit and cranium in the region of the optic foramen with injury to the nerve was removed at operation.

H. H., white boy fourteen years of age, consulted us December 7, 1930. He gave a history of having stuck a stick in the right orbit when he fell from a tree the day before. Following this injury, he complained that he could not see out of the right eye.

On examination, a small wound in the right upper lid about 1 cm. from the inner canthus was to be seen. There was a marked exophthalmos, the globe was immobile and the pupil was widely dilated. Even though the appearance of the disc and vessels was normal, the patient had no light perception. Ptosis was complete.

The patient was x-rayed by Dr. Dalton Richardson. The picture revealed a small hole in the posterior table of the orbit. No foreign body could be detected on the plate.

The patient was quite ill and was immediately hospitalized. His temperature ran a hectic course, and he complained of headache and was quite drowsy. On December 12, he was taken to the operating room, the wound was opened, and a stick of wood 2 cm. x 3 mm. was removed, under general anaesthetic. At the time of operation, we were not satisfied with our findings but his general condition was such that we could do no more exploring. He was returned to his room in good condition. He improved and was permitted to go home on the 23rd of December for Christmas.

(Hilgartner). X-ray of orbital injury.

On January 7, 1931, he returned. There was an open sinus draining a large amount of pus through the original wound. His general condition was much improved. Under local anaesthetic, the wound was again opened, but nothing was found.

Because of the meningeal symptoms that he showed following the injury, we were afraid to do too much manipulating for fear of starting up some trouble and decided to follow the safest course, allowing more time for a thorough walling off of the foreign body which we suspected was still present.

On February 5th the wound was opened again under local anaesthetic, and a stick of oak 6 cm. x ½ cm. was pulled out.

The wound healed immediately and the patient was discharged on February 9.

At the time of discharge the patient showed the following changes: 1. complete ptosis of right upper lid, 2. exophthalmos, 3. internal strabismus, 4. very slight movement of globe, 5. no light perception, 6. an atrophied nerve head, and 7. dilatation of pupil.

On comparing the x-ray and a disarticulated skull, we were able to ascertain that the stick had pierced the orbit

and passed slightly upward, going through the optic canal and sectioning the optic nerve on the same side.

The left nerve was not involved at all.

209 Norwood building.

PARATHYROID CATARACT

M. E. MARCOVE, M.D.
DENVER

A case is reported in which lenticular opacities developed as symptoms of tetany appeared in a patient following thyroidectomy. Under adequate treatment with calcium and parahormone the symptoms disappeared and the lenses showed no further change. Reported to the Colorado Ophthalmological Society, February, 21, 1931.

It has long been known that cataracts often occur in parathyroid tetany. This association has been confirmed numerous times by animal experimentation. The mechanism by which these cataracts are developed has not been sufficiently worked out, for cataracts are rarely seen in tetany of other than parathyroid origin. Neither the alkalosis nor the disturbance in calcium metabolism seems to be responsible for the disease, though the latter has been assumed by some authors. Complete removal of the parathyroid glands is not necessary as sometimes the glands are injured by the grasp of a hemostat or by some direct interference with their blood supply.

In human beings, about forty cases of cataract following thyroidectomy have been reported. The following is a report of another case: W. H. D., male, aged 41 years, was first seen in October, 1929, with the following history. Early in 1925 he developed an exophthalmic goiter for which he was operated on in July, followed by subsequent operations in August and September of the same year. He felt better for almost one year after these operations, but, the same type of symptoms returned. He was treated medically but received no relief, and was again operated on, on October 14, 1927. Ten days after the operation the patient showed signs of parathyroid tetany. The chief symptom at this time consisted of tingling of the skin in the extremities. This complaint persisted until the middle of November, 1927, when he began to have severe pain in the extremities and chest, with typical carpopedal spasms. H was given calcium and desiccated parathyroid gland by mouth without relief. Five per cent calcium chloride orally gave immediate relief, which lasted for three days. He improved in every way for about a month after which the muscular spasms returned, and were not influenced by treatment similar to that used before, but were relieved by intravenous calcium. The patient lost all his hair, and his fingernails became brittle. The serum calcium was decreased, and the inorganic serum phosphorous was increased.

Fifteen months after the operation the patient complained of failure of vision in both eyes, more marked in the left. There were no other ocular disturbances, the vision at this time being O.D. 20/100, O.S. 20/200; with correction, O.D. 20/70, O.S. 20/200. External examination was negative, the lenses were cloudy, with the left more involved. The fundi were seen through a haze, but no pathology was made out. Biomicroscopic examination showed the opacities of the lens to be very fine, and to be located exactly under the anterior and posterior capsules. Upon higher magnification, the opacities were seen to be composed of very fine vacuoles which were located chiefly in the pupillary area, and were most pronounced just beneath the posterior capsule. Both eyes were similar, except that the left was more advanced. The patient's refraction was measured and with —0.25sph. —1.50 cyl. axis 165°

vision O.D. equalled 20/20; with —1.75 sph. —0.37 cyl. axis 165° vision O.S. equalled 20/40+. He was given a two per cent dionin solution. General treatment at this time consisted in daily injections of thirty units of parahormone and 300 grains of calcium lactate by mouth. The subsequent history over a period of sixteen months shows the patient to have been entirely free from convulsive seizures. His hair gradually returned, nails became normal, and the eyes changed very little, except that the opacity of the left lens in the posterior cortex became a little more dense. The vision at this time was O.D. 20/20—; O.S. 20/70+, with correction.

Goldmann carried out researches on rats in which he deprived the animals of their parathyroids. He reports that cataracts develop in these animals in from three to six months, but only in those animals that have muscular symptoms. If, by appropriate medication, muscular symptoms are avoided, the lenses remain clear. Several hours after a muscular crisis, tiny subcapsular vacuoles appear in the form of a comma arranged in the direction of the lens fibers. If there is no new crisis, these vacuoles may disappear entirely. If there are further crises, these vacuoles increase in number, and arrange themselves according to the lens fibers. Gradually the whole affected layer is buried in the depth of the lens, and its place is taken by new transparent fibers.

Pellathy examined the lens microscopically in an animal whose parathyroids had been removed. The opacities found were always subcapsular. Between the lens fibers were cavities filled with drops of albumin or other homogeneous substance. In the superficial cortical layers, these formations were fine. In the deeper layers, they became coarse and confluent. The nucleus and the ciliary epithelium remained unchanged.

No satisfactory explanation has yet been put forward to account for the lenticular changes. Greenwald cites evidence to support the fact that in the absence of the parathyroid hormone, there is an extensive deposit of calcium in the tissues, and that the cataracts observed after parathyroidectomy may be due to a deposition of this salt in the lens.

This case shows the value of a proper treatment for parathyroid deficiency on the development of lenticular opacities. The fact that in over sixteen months these cataracts have practically remained stationary shows that the cause of progression in the lens is due to a tetanic crisis, which if controlled, also controls the pathological process of the lens.

526 Republic building.

VARIATIONS OF THE NORMAL BLIND SPOT WITH SPECIAL REFERENCE TO THE FORMATION OF A DIAGNOSTIC SCALE

I. The Form Blind Spot

HAZEL A. WENTWORTH, PH.D.
PHILADELPHIA

A study of the blind spots of 200 normal persons is made under conditions standardized as to illumination, size of test object, light adaptation and method of mapping. Results are recorded and tabulated according to area and position. The areas varied from 6.8 sq. cm. to 17.5 sq. cm., the average being 11.4 sq. cm. at 33 cm. from the eye, using a one degree test object. The average size was found to increase somewhat with age. The distribution curve rather than the average size is regarded as of most significance as a standard for diagnostic comparisons. The author feels that the series is too small for generalization. From the Department of Ophthalmology, School of Medicine, University of Pennsylvania.

From the standpoint of practical perimetric work in office and clinic, it is important to know not only the average size, shape and position of the blind spot but also the variation to be expected from eye to eye in normal, as well as pathologic conditions of the eye. Without this knowledge diagnosis is impossible. It is important, also, if changes in size and shape of the blind spot are to have any value as an index of early pathologic changes, that the amount of variation should be small for non-pathologic eyes relative to that produced by pathologic conditions.

The progress of pathologic conditions as shown by changes in the size and shape of the blind spot can be determined with an acceptable degree of accuracy. That is, through a control of all possible variable factors, a satisfactory degree of precision and reproducibility can be obtained in repeated examinations of any single nonpathologic eye. While the influence of certain of these variable factors has not been completely determined in case of the blind spot, these factors are similar in many instances to those which influence the size and shape of the visual field. Like the latter, also, they are more numerous and have greater effect upon the blind spot for colored than for colorless, or form stimuli. The color blind spots and the factors pertaining to them will be discussed at a later date. The possible factors which should be controlled in any determination of the form blind spot may be stated as follows: Intensity of stimulus or test object (dependent upon the intensity of illumination when pigment papers are used); size or visual angle of the test object, length of exposure to the eye, method of exposure (moving or stationary test object), method of approach (from blind to seeing area or vice versa), intensity of general illumination and consequent state of adaptation of the retina, and brightness of preexposure and surrounding field or background. All of these may be controlled and standardized.

Those factors, on the other hand, that influence the results for different eyes, such as age, curvature of the cornea, errors of refraction of both central and peripheral field, sensitivity of the retina and the distribution of that sensitivity over the retina, are for the most part innate and not amenable to control. It is the variation due to these factors, which must be allowed for in diagnosis. A method suggested for making this allowance is that of a diagnostic scale showing the distribution of results to be expected, first for non-pathologic and eventually for pathologic conditions of the eye, this scale to be based on the results from a large number of cases sampled so broadly as to include as many as possible of the factors the influence of which cannot be eliminated. The value of such a scale has been pointed out a number of times. Briefly, it provides a method of separating the pathologic from the nonpathologic cases. That is, a means is provided for placing each new case in a group the rating of which with regard to the distribution of normal cases is known. It thus provides a fixed objective standard

Table I

SHOWING THE MEASUREMENTS OBTAINED FOR THE NONPATHOLOGIC BLIND SPOT AND THE CONDITIONS UNDER WHICH THEY WERE OBTAINED BY DIFFERENT INVESTIGATORS.

Investigator	Young	Hannover and Thomsen	Griffin	Listing	Fick and Du Bois Reymond	Aubert	Wittick	Landolt	Bull	Bjerrum
Date	1801	1832	1838	1853	1853	1865	1863	1873	1881	1890
No. Cases		22	11		2	1				
Ages										
Size and Type of Stimulus	candle flame	blackened pen point	dark paper*		small circle black paper			white		1–20 mm. ivory balls
Distance Eye from Screen	16 feet		across room		3 meters			perimeter radius	30 cm.	2 meters
Type of Screen		white paper	light colored wall		bright gray wall	Foerster perimeter?		various perimeters	Foerster perimeter	black velvet curtain
Illumination					well lighted room					"good daylight"
Fixation	candle flame	mark on paper			small circle black paper			light thru 3.5 × .15 cm. aperture		
Method of Determining Limits			moved eye until stimulus disappeared							

Measurements:

Area:	Av.									
	Range									
Breadth: (horizontal meridian)	Av.	3°5′	6°10′	7°31′†	5°55′9″	5°33′33″	5°51′	7°45′	6°45′	6°30′	5°4′48″
	Range		3°39′–9°47′			5°24′–6°18′					
Length:	Av.						6°				
	Range										
Center to Fixation:	Av.			15°34′							
	Range			15°4′–16°18′							
Center below Horizontal:	Av.			1°22′							
	Range			1°.5′–1°17′							
Center to Upper Border:	Av.										
	Range										
Center to Lower Border:	Av.										
	Range										
Fixation to Nasal Border:	Av.	12°56′	11°56′		12°37′5″	12°27′	14°21′	14°30′			
	Range	18°	9°58′–14°27′			12°–12°54′					
Fixation to Temporal Border:	Av.	16°1′	18°		18°33′24″	18°18′	20°34′				
	Range		15°29′–21°43′								

* Used also: 1. image of light in mirror—†6°27′ 2. luminous point thru cylinder—6°6′ 3. two unshaded candles—2°45′

Table 1—continued

Investigator	Helmholtz	Van der Hoeve	Peter	Gradle	Eichenberg	de Vincentiis	Ferree, Rand and Wentworth	Traquair	Incze	Wentworth
Date	1896	1912	1916	1916	1921	1922	1925	1927	1929	1931
No. Cases		100	"several hundred"	13	184	18	10		66	200
Ages		18–22			14–20 (72 cases) 20–50 (20 cases)		18–22		20–26	14–55
Size and Type of Stimulus	inked pen point	1–1.5 cm.		3 mm. blue steel balls	5 mm. white	5–10 mm.	(1) 2.5 mm. white (2) 2.5 mm. black	10–20 mm.	(a) 5 mm. (b) 1–1.5 cm.	5.8 cm. white = 1°
Distance Eye from Screen	8–12 inches	2 meters	16.5 cm.	60 cm.	2 meters	120 cm.	33 cm.	2–4 meters	(a) 1 meter (b) 2 meters	33 cm.
Type of Screen	white paper	black campimeter screen	black hand campimeter	white celluloid solenoid	black screen	black screen	(1) black (2) white screen*		blackboard	black tangent screen*
Illumination						bright daylight	7 ft.c. artificial daylight			7 ft.c artificial daylight
Fixation	cross mark			double fixation device-parallax principle	5 mm. round white object	bright point on screen	peep-sight parallax device*		white mark on board	peep-sight parallax device*
Method of Determining Limits		blind to seeing area		ball moved by magnet			Av. of limits for blind to seeing and seeing to blind areas		(1) blind to seeing area (2) seeing to blind area	blind to seeing area

Measurements:											
Area:	Av.							(1) 8.43 sq. cm. 5.7—11.6 sq. cm. (2) 9.06 sq. cm. 6.7—13.0 sq. cm.			11.4 sq. cm. 6.8–17.5 sq. cm.
	Range										
Breath: (horizontal meridian)	Av.	6°56′	5°42′55″ 4°43′ 3″– 6°32′45″	5°28′	4°54′	3°46′ 1°–6°33′	5°30′	(1) 4°28′36″ (2) 4°28′36″	5° 7′5″	(1) 6°14′37″ (2) 4°45′27″	5°29′36″ 4° 9′18″– 7°25′18″
	Range										
Length:	Av.		7°26′ 6°19′32″– 8°56′41″	7°49′	7°45′	5°28′ 4°–7°	7°15′	(1) 6°45′36″ (2) 7°14′52′	7°17′	(1) 7°39′33″ (2) 6°51′54″	7°45′ 6°3′–9°25′26″
	Range										
Center to Fixation:	Av.		15°33′47″ 12° 1′28″– 18° 0′15″	15°49′	16°33′32″	16°30′ 14°37′–18°6′	15°	(1) 16°30′ (2) 16°30′ 14°30′–17°30′	15°37′36″		16°10′ 12°33′18″– 18°44′20″
	Range										
Center below Horizontal:	Av.		1°40′41″ 0° 5′34″– 4°58′20″	1°30′	0°54′30″*	1°25′*	"Sometimes as much as 3°"		1°24′		1°29′6″
	Range										
Center to Upper Border:	Av.			2° (approx.)	2°58′	0°36′–3°18′					2°23′24″ 0°–5°5′24″
	Range										
Center to Lower Border:	Av.			5° (approx.)	4°47′						5°21′36″ 2°42′36″– 8°29′24″
	Range										
Fixation to Nasal Border:	Av.	12°25′			13°15′35″						13°36′54″ 9°48′– 18°25′54″
	Range										
Fixation to Temporal Border:	Av.	18°55′			18° 9′35″						17°48′24″ 14°55′48″– 21°12′
	Range										

* Calculated from data given

* Special attachments for Ferree-Rand Perimeter

* Special attachments for Ferree-Rand Perimeter

for comparison in place of the variable standard subjectively determined by each physician according to his experience and judgment, an obvious handicap to the younger man in the clinic, and makes possible an accumulation of comparable data from a number of sources which can be made available to all using the same procedure. It is obvious, of course, that a scale of this type could be applicable to any given case only when the examination had been made under identical conditions with those for which the scale was made.

A number of investigators have attempted to determine the average size, shape and position of the normal blind spot with widely varying results, due, admittedly, to a failure to observe a standard set of conditions. Size of test object used, its distance from the eye, the relative brightness of background and test object, control of fixation, methods of mapping and the general illumination under which the work was done (seldom even stated) all varied from person to person. No real comparison of their results is, therefore, possible. A summary of the results of every available investigation of the normal blind spot and the conditions under which they were obtained is given in Table I. In the earlier studies, the results are meager, and are concerned largely with the position and horizontal diameter of the blind spot as determined on the perimeter arc. Following Bjerrum's development of the tangent screen more detailed studies were made and averages obtained for a sufficiently large number of observers to be considered authentic. Little attention was paid, however, to the range of value obtained in a group of observers. Van der Hoeve mentions the limits of variation. Eichenberg alone gives the actual distribution of measurements. He was handicapped, however, in having no single index for the size of blind spot obtained. He presented separate distribution curves for the horizontal and vertical diameters, distance of the center of the blind spot from the point of fixation and distance to which the blind spot extended above the horizontal line passing through fixation. As no one of these measurements or any combination of them so far as the writer has been able to discover, is a reliable index of the actual size of the blind spot in any given case, it is difficult to apply his results to practical work even were the conditions of working which he observed such as to give a high degree of precision and reproducibility in repeated examinations.

In all types of field study, as pointed out in a previous paper, a comparison can be made with the minuteness and precision necessary for laboratory and diagnostic purposes only when a single value or index is to be had which can be taken as fairly representative of the retinal area under investigation. In the case of the blind spot, the most accurate and convenient index of the actual extent of the insensitive area to be found, perhaps, is the area of the plotted graph, provided the limits of sensitivity have been determined and plotted in a sufficient number of radial lines. In our own work this area has been measured by means of a planimeter. This measurement is extremely quick and simple and the instrument is readily available to any office or clinic.

It has been our purpose in the present study to show the variations in the form blind spot under conditions comparable to those met with in ordinary clinic and office practice, obtained in a sufficient number of non-pathologic cases to serve as a nucleus for a diagnostic scale such as we have described.

Conditions under which the work was done. The experimental work was done on the Ferree-Rand perimeter, the advantages of which for perimetric and scotometric work have been stated a number of times in the literature.

The special features of this instrument which are of particular value for blind spot study are the provisions for the control of intensity and color of light and for securing evenness of illumination; the devices and adjustments for the accurate location of the eye at the center of the perimetric system and for securing steady and precise control of fixation; and the special at-

tachments for blind spot and scotometric determinations providing for the control of brightness of pre-exposure, surrounding field, and other variable factors.

The attachment found most convenient for practical scotometric and blind spot work is that of a small tangent screen of 30° radius. This screen is attached to the perimeter carriage by means of a hollow shaft which slides into the tubular aperture about which the perimetric arm rotates. When locked in position, i.e., with its central point in the axis of rotation of the perimetric arm, the tubular opening of the perimeter is continued through to the front surface of the tangent screen. This makes possible the exact adjustment of the screen as well as the use with it of all of the fixation controls provided for the perimeter. Detachable slates of thin hard sheet aluminum covered with washable material of black, white or grays of the brightness of the colors used, are provided. On these the fields are laid off on the tangent scale. Faint radial lines at 15 degree intervals and concentric circles at 5 degree intervals with the approximate outlines of the average normal blind spot for both eyes provide convenient points of reference for transferring the tracings obtained to permanent charts that may be filed. In practical work this screen is preferable to the special attachment provided for blind spot determinations because of the speed with which not only the blind spot but scotomata may be found and outlined and accurately located in relation to each other and to the entire field of vision of either eye.

In determining the limits of the blind spots two methods of approach are commonly used. The test object may be passed outward from the insensitive to the sensitive visual area of the retina or vice versa. Gradle found that the area of the blind spot obtained by the two procedures differed in size but stated that this was of little practical consequence provided a standard procedure was adopted and adhered to. He used the first method. Incze, also, found the blind spot larger when the stimulus was passed from within the blind spot outward than when the reverse method was used but concluded from results obtained by placing stationary objects in the area between the two outlines and suddenly exposing them that the larger blind spot was the true one.

In previous work by the writer as in that of Ferree and Rand both procedures have been used and the average taken. With accurate observers the limits were found to coincide. When they did not the percentage of error was found to be very low after a little practice, the variation not exceeding the width of the test object used, or slightly less than 0.5 of a degree. This work was done in a college laboratory with students who were young, alert, intelligent and trained in laboratory procedure. In clinic, hospital or office, however, where there is little opportunity for practice and the patients are untrained, often ignorant, unintelligent and in mental or physical distress or both, we have found a much greater reliability and reproducibility of result when the test object is passed from the insensitive area outward until just visible than when the test object is first placed in an area sensitive to visual impression and moved toward the blind spot or even when the average of the two is taken. This is due undoubtedly to the difficulty of maintaining a steady fixation while attempting to observe an object elsewhere in the field of vision, a task, even for the trained observer. If the test object is within view the eye tends to follow it, thus reducing the apparent size of the blind spot. If it is within the blind or insensitive area, fixation is more easily maintained until it becomes visible. The point of appearance is therefore fairly accurately determinable even though the patient is unable to prevent a slight shift in fixation at the moment the object comes into view.

In determining the limits of the blind spot for nonpathologic cases, therefore, the position of the blind spot was first located by passing the test object along the horizontal meridian, passing through the point of fixation until the observer reported its disappearance and then reappearance on the further side

of the blind spot. The blind spot thus approximately located, the test object was passed radially from the center toward the outer edge of the blind spot until the observer reported its appearance. This position was marked and the procedure repeated at sufficiently close intervals to give an accurate outline of the blind spot.* Generally, 16 to 24 points were sufficient. In order that the reliability of the results might be determined both methods of approach were occasionally used and the points of both the appearance and disappearance of the test object noted. In the majority of cases they were found to coincide, when they did not the difference seldom exceeded 0.5 degree. Where any difference in the point of appearance and disappearance was found the former was taken as the limit after being carefully checked.

The test object consisted of a white disc, 5.8 mm. in diameter, mounted on a slender black rod. This subtends a visual angle of one degree at 33 cm., the distance of the screen from the eye. This is somewhat larger than was previously used by the writer but has been found the most satisfactory and generally applicable in routine work. The screen used was black. The intensity of illumination of the tangent screen was kept constant at seven foot candles. This constancy was maintained through ammeter and rheostat control of the current operating the lamp used to illuminate the arc of the perimeter. At the beginning of each working period the sensitivity of the eye was standardized by a ten minute period of adaptation to the illumination used. A part of this time was used in making adjustments and locating roughly the center of the blind spot. Sufficient rest periods were allowed to guard the eye against fatigue. While the results were carefully checked no previous practice was given for, as we have stated, it was our purpose to determine norms for conditions comparable to those which obtain in ordinary office and clinic practice where diagnosis must often be made from a single examination of a person untrained and unfamiliar with tests of this character.

Two hundred blind spots were obtained. The cases examined included nurses, assistants on the hospital staff, a large number of clinic patients and a few ambulatory patients from the surgical wards. No case was included which showed the slightest evidence of eye pathology or systemic conditions which might presumably affect changes in retinal sensitivity.

Results: The results are given in detail in Tables II and III, and are shown graphically in Figs. 1 and 2. The areas of the blind spots are given in Table II, the average and total range of values in Table III. The areas were measured by means of a planimeter and represent the size of the blind spot at a distance of 33 cm. from the eye. A graphic representation of these areas and their distribution is given in Fig. 1. The total ranges of sizes for 200 eyes was from 6.8 to 17.5 sq.cm. For convenience of representation in plotting, these were divided into groups, each of which covered a range of 1.5. sq. cm. The number of cases in each group is represented on the vertical coordinate, the range in size on the horizontal coordinate. Separate distributions are given for persons under 25 years of age and over 25 years of age respectively. In Fig. 2, blind spots have been plotted to show the area of the smallest blind spot, the largest blind spot, the average blind spot for 200 eyes and the size of blind spot considered borderline or suspicious. These are shown schematically in (a); as they actually were found, in (b).

A comparison of the results with those of previous investigators is given in Table I. For the sake of comparison also, the measurements of length, breadth and position of the blind spot in relation to the center of the field have been included in Table III.

* Curiously enough, this same change in method of determining the limits of the blind spot or other scotomas has been made by Ferree and Rand as noted by them in an article published since the writing of the present paper. See Ferree, C. E., Rand, G. and Sloan, L. L., Sensitive Methods of the Detection of Bjerrum and other Scotomas, Arch. of Ophth., 1931 (Feb.), v. 5, pp. 224-260.

Table II

SHOWING THE INDIVIDUAL MEASUREMENTS OF 200 NONPATHOLOGIC BLIND SPOTS (ODD NUMBERS SHOW MEASUREMENTS FOR RIGHT EYE; EVEN NUMBERS, FOR LEFT)

Case No.	Area	Breadth (horizontal meridian)	Maximum Breadth	Length	Distance of center from fixation	Case No.	Area	Breadth (horizontal meridian)	Maximum Breadth	Length	Distance of center from fixation
	Sq. cm.	mm.	mm.	mm.	mm.		Sq. cm.	mm.	mm.	mm	mm.
1	12.5	36	37	40	92.0	51	10.6	18	32	45	85.0
2	10.6	32	34	43	90.0	52	9.9	28	30	42	86.0
3	12.5	28	34	52	92.0	53	9.1	22	28	42	94.0
4	12.9	34	36	50	86.0	54	10.6	29	31	46	90.5
5	6.8	24	27	38	89.0	55	11.4	17	34	50	91.5
6	10.6	23	30	48	89.5	56	13.7	20	32	47	88.0
7	12.2	32	32	50	89.0	57	12.2	29	32	48	92.5
8	14.8	33	36	54	89.5	58	14.4	33	34	50	94.5
9	10.6	30	31	49	92.0	59	7.6	23	25	38	91.5
10	10.6	26	32	48	89.0	60	10.3	26	26	39	88.0
11	11.4	22	30	52	90.0	61	9.1	25	26	39	93.5
12	9.5	28	32	44	92.0	62	9.9	26	28	42	100.0
13	11.0	29	30	44	87.5	63	14.0	37	40	47	91.5
14	7.6	24	26	28	88.0	64	14.0	31	33	56	97.5
15	12.2	32	34	47	92.0	65	17.5	43	44	41	106.0
16	12.5	36	36	52	98.0	66	14.4	32	35	48	106.0
17	11.4	22	29	46	93.0	67	11.0	30	30	43	95.0
18	12.9	22	30	50	88.0	68	13.7	30	32	42	83.0
19	11.0	30	32	46	91.0	69	12.5	0	32	48	98.0
20	12.9	35	33	52	91.5	70	14.4	30	34	56	93.0
21	10.3	24	28	46	92.0	71	9.5	29	35	39	91.5
22	11.8	25	29	54	89.5	72	9.5	27	29	43	92.5
23	9.1	26	26	41	83.0	73	15.2	33	36	52	73.5
24	11.2	33	30	43	84.5	74	12.5	31	32	50	80.5
25	15.2	35	36	52	93.5	75	13.3	35	35	48	95.0
26	10.6	29	32	44	89.5	76	11.4	6	36	42	87.0
27	11.4	32	34	46	88.0	77	14.0	35	36	52	89.0
28	11.4	28	32	44	92.0	78	15.2	34	34	52	96.0
29	10.3	27	30	43	93.5	79	11.4	33	34	50	88.5
30	13.3	30	34	49	94.0	80	9.9	25	27	42	94.5
31	13.7	33	33	51	93.5	81	12.5	31	34	46	81.5
32	15.6	35	35	50	92.5	82	12.5	31	34	48	86.5
33	10.6	27	27	37	102.5	83	11.4	30	30	44	95.0
34	8.0	27	28	41	109.5	84	13.3	35	35	52	97.5
35	14.8	31	32	52	91.5	85	7.2	23	25	36	92.5
36	11.8	32	34	50	94.0	86	7.6	6	28	42	102.0
37	15.2	32	34	50	91.0	87	11.4	25	27	42	93.5
38	11.4	30	31	40	92.0	88	12.2	30	33	47	90.0
39	8.8	25	25	37	93.5	89	12.2	31	32	44	86.0
40	9.5	26	29	42	92.0	90	9.9	30	32	42	85.0
41	9.1	24	26	43	90.0	91	13.3	32	34	45	96.0
42	10.6	27	32	44	90.5	92	12.5	31	33	46	94.5
43	10.3	24	24	41	88.0	93	9.5	28	30	42	94.0
44	10.6	30	31	44	88.0	94	11.4	9	33	44	103.5
45	10.6	29	30	43	92.5	95	9.9	31	30	43	93.5
46	12.9	32	33	48	93.0	96	10.3	29	32	44	91.5
47	10.3	30	31	39	93.0	97	12.2	10	32	50	92.0
48	11.4	27	31	43	93.5	98	10.6	23	29	46	91.5
49	10.3	30	31	52	98.0	99	11.8	31	32	44	93.5
50	11.4	29	32	48	89.5	100	11.4	32	32	48	94.0

Table II (cont.)

SHOWING THE INDIVIDUAL MEASUREMENTS OF 200 NONPATHOLOGIC BLIND SPOTS (ODD NUMBERS SHOW MEASUREMENTS FOR RIGHT EYE; EVEN NUMBERS, FOR LEFT)

Case No.	Area	Breadth (horizontal meridian)	Maximum Breadth	Length	Distance of center from fixation	Case No.	Area	Breadth (horizontal meridian)	Maximum Breadth	Length	Distance of center from fixation
	Sq. cm.	mm.	mm.	mm.	mm.		Sq. cm.	mm.	mm.	mm.	mm.
101	11.4	33	33	46	88.5	151	11.4	29	33	48	91.5
102	10.6	28	32	46	90.0	152	12.9	32	34	50	93.0
103	11.4	27	27	47	93.5	153	10.6	30	32	48	91.0
104	8.8	24	25	42	92.0	154	11.8	36	36	48	91.5
105	9.5	29	29	37	98.5	155	9.9	32	32	46	91.0
106	9.5	18	33	47	104.0	156	11.0	30	33	44	93.0
107	15.2	34	38	54	91.0	157	11.0	31	35	44	94.5
108	14.0	30	33	50	91.0	158	11.4	23	29	51	92.5
109	11.4	30	30	45	93.0	159	12.2	31	32	48	94.5
110	10.6	29	30	45	96.5	160	8.0	24	27	42	112.0
111	8.0	25	26	39	90.5	161	11.0	30	30	45	95.0
112	11.0	30	32	40	91.0	162	9.5	25	27	44	91.5
113	10.3	26	29	45	89.0	163	9.9	27	31	41	92.5
114	13.7	36	36	50	92.5	164	8.4	24	27	39	91.0
115	8.8	27	28	45	86.5	165	8.8	27	29	42	87.5
116	9.1	28	28	39	83.0	166	8.0	22	29	38	88.0
117	14.4	31	36	54	92.5	167	14.4	36	36	50	94.0
118	16.0	38	38	54	94.0	168	13.3	29	35	42	92.5
119	12.2	32	33	46	97.0	169	7.2	26	27	36	85.0
120	11.8	32	32	48	95.0	170	8.8	23	28	41	90.5
121	9.9	19	29	39	87.5	171	11.4	31	34	46	93.5
122	11.0	31	30	43	90.5	172	13.3	22	36	51	91.0
123	11.0	21	30	46	90.5	173	14.0	33	35	52	93.5
124	11.4	28	32	47	94.0	174	13.3	34	34	54	87.5
125	9.9	27	29	41	92.5	175	10.6	31	31	46	96.0
126	8.4	26	29	38	93.0	176	12.2	30	31	49	95.0
127	12.5	32	32	48	92.0	177	12.2	32	34	49	92.0
128	15.6	30	35	55	91.0	178	15.2	34	35	50	94.0
129	11.8	32	32	52	93.0	179	9.5	24	29	43	92.0
130	12.9	24	32	52	90.0	180	11.4	30	35	46	96.0
131	12.2	30	31	46	93.0	181	11.4	31	33	45	93.0
132	13.7	34	35	54	96.0	182	10.6	26	33	40	92.0
133	10.6	32	32	48	88.0	183	12.2	34	34	47	94.0
134	11.4	36	37	47	88.5	184	11.0	27	34	45	100.5
135	14.4	34	37	52	95.0	185	11.0	30	30	46	96.0
136	16.3	33	37	56	93.5	186	9.0	24	27	43	90.0
137	14.0	36	36	50	94.0	187	8.8	26	29	42	89.0
138	14.0	34	34	48	88.0	188	8.8	27	28	40	90.5
139	11.4	30	32	43	95.0	189	13.7	36	36	50	90.0
140	9.9	26	30	43	92.0	190	11.4	27	33	44	88.5
141	12.5	33	33	49	95.0	191	6.8	24	25	36	92.0
142	13.7	32	36	50	93.0	192	7.6	23	26	39	93.5
143	10.6	29	31	45	81.5	193	9.5	27	27	40	90.5
144	11.0	31	32	42	88.5	194	8.8	26	28	40	88.0
145	11.8	33	34	46	94.5	195	13.3	32	32	51	91.0
146	11.4	32	32	48	92.0	196	11.4	34	34	46	93.0
147	8.8	10	27	41	83.0	197	10.3	28	28	44	96.0
148	8.0	16	27	40	89.0	198	12.2	27	36	42	89.5
149	10.6	27	29	45	93.5	199	13.3	29	34	50	91.5
150	10.3	26	27	44	92.0	200	10.3	29	30	44	91.5

Table III

SHOWING THE AVERAGE AND RANGE OF MEASUREMENTS FOR 200 NONPATHOLOGIC BLIND SPOTS

Blind Spot Measurements	Average		Range		Mean Deviation		Maximum Deviation	
	cm.	degrees	cm.	degrees	cm.	degrees	cm.	degrees
Maximum Breadth	3.16	5.49	2.4– 4.3	4.16– 7.32	0.30	0.57	1.15	2.00
Breadth along horizontal diameter of field	2.83	4.92	0– 4.4	0 – 7.42	0.47	0.82	2.83	4.92
Length	4.56	7.75	3.6– 5.6	6.05– 9.59	0.38	0.61	1.04	1.67
Distance from fixation point to center	9.19	16.17	7.4–11.2	12.56–18.74				
Distance from fixation point to nasal border	7.77	13.62	5.7–10.0	9.80–18.13				
Distance from fixation point to temporal border	10.60	17.81	8.8–12.8	14.93–21.02				
Distance of upper border above horizontal meridian: all cases	1.41	2.39	0– 3.0	0 – 5.09				
92% cases			0.8– 2.0	1.35– 3.39				
75% cases			1.0– 1.8	1.69– 3.04				
Distance of lower border below horizontal meridian: all cases	3.15	5.36	1.6– 5.0	2.71– 8.49				
75% cases			2.7– 3.7	4.48– 6.22				
Distance of center below horizontal meridian	2.28	1.49						
	sq. cm.		sq. cm.		sq. cm.		sq. cm.	
Area	11.4		6.8–17.5		1.6		5.4	

The results may be summarized as follows:

1. The average distance of the center of the blind spot from the center of the field of vision was 16.17 degrees, the range 12.56 to 18.74 degrees.

2. The average distance of the center of the blind spot below the horizontal meridian of the field was 1.49 degrees.

3. The greatest average breadth of the blind spot was 5.49 degrees, the range 4.16 to 7.32 degrees. The average breadth in the horizontal meridian of the field was 4.92 degrees, the range 0 to 7.42 degrees. The mean variation was 0.57 degree and 0.82 degree respectively.

4. The average length of the vertical axis of the blind spot was 7.75 degrees; the range, 6.05 to 9.59 degrees; the mean deviation, 0.61 degree.

5. The average distance of the upper border of the blind spot from the horizontal or 180° meridian of the field was 2.39 degrees; the range from 0 to 5.09 degrees. Ninety-two per cent of the blindspots were not over 3.39 degrees or less than 1.35 degrees above the horizontal; three were entirely below the horizontal meridian.

6. The average distance of the lower border of the blind spot from the horizontal meridian of the field was 5.36 degrees; the range from 2.71 to 8.49 degrees. Seventy-five per cent of the cases were not more than 6.22 degrees and not less than 4.48 degrees from the horizontal.

7. The average distance of the nasal border of the blind spot from the center of the field along the horizontal meridian was 13.62 degrees; the range from 9.8 to 18.13 degrees.

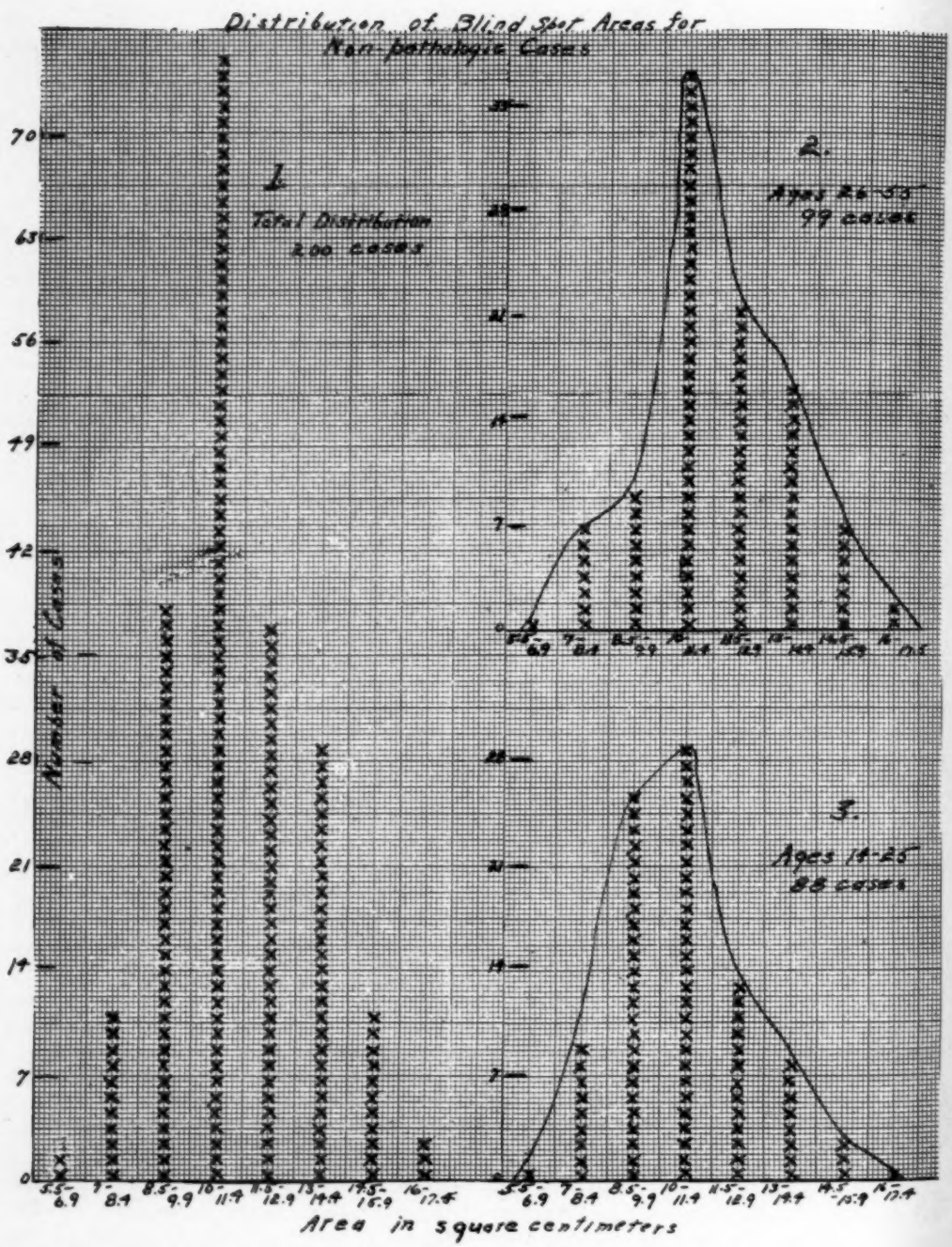


Fig. 1 (Wentworth). Showing the distribution of (1) 200 nonpathologic blind spots; (2) those cases over 25 years of age; (3) those cases of 25 years of age or under.

8. The average distance of the temporal border of the blind spot from the center of the field along the horizontal meridian of the field was 17.81 degrees; the range from 14.93 to 21.02 degrees.

9. The average size of the blind spot as measured by the planimeter was 11.4

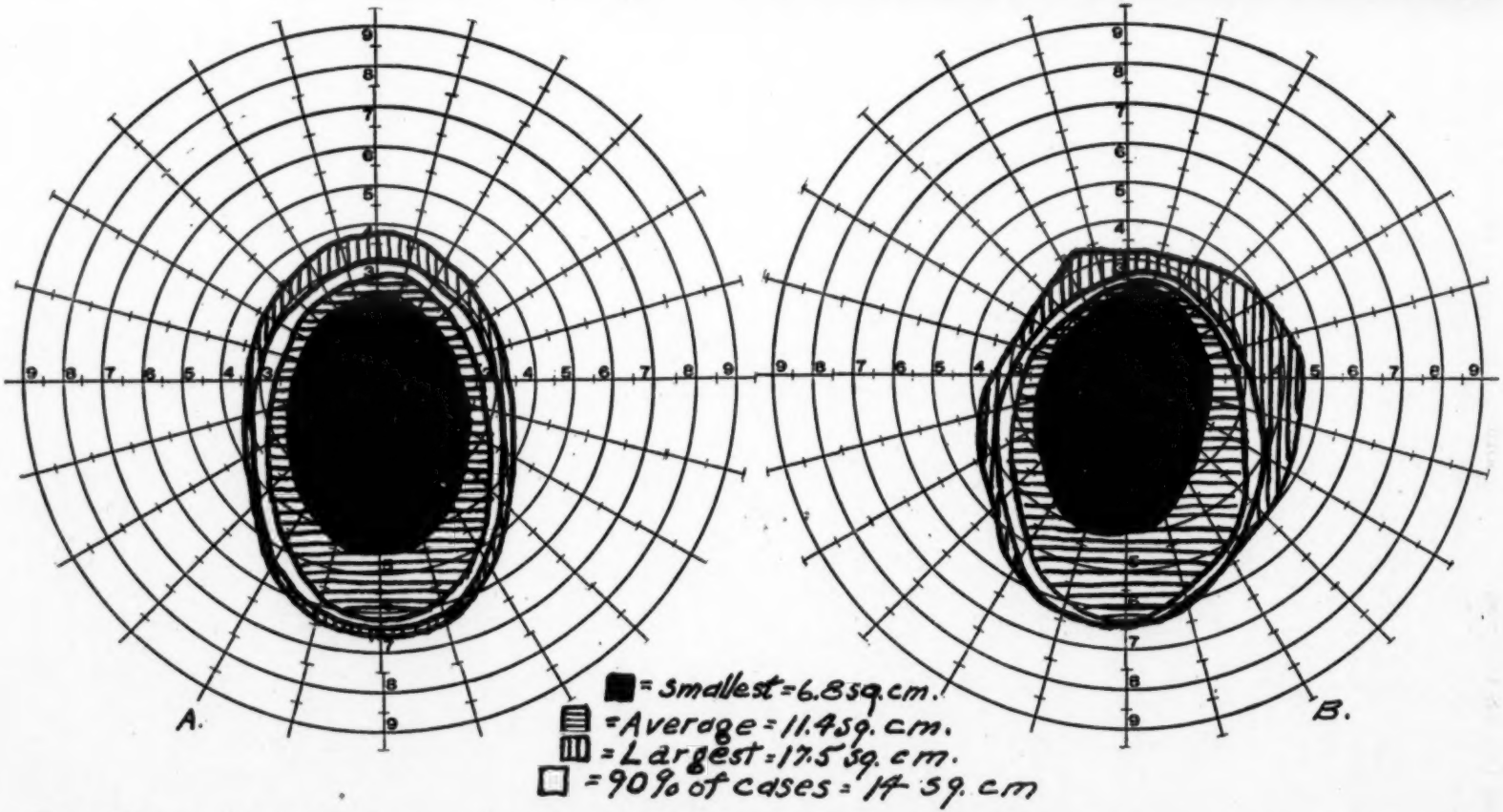


Fig. 2 (Wentworth). Showing the average, smallest and largest areas of 200 nonpathologic blind spots for form (1° white stimulus on black background), also the area including 90% of cases. A: Schematic diagram. B: Blind spots actually obtained.

sq. cm.; the range from 6.8 to 17.5 sq. cm. with 92 per cent of the cases less than 14 sq. cm. in area. The average deviation was 1.6 sq. cm.

10. The vertical axis of the blind spot was perpendicular in each eye for only 10 of the 100 persons examined. For 19 persons, the axis was perpendicular in one eye, that of the blindspot in the other eye converging toward it in 7 instances; diverging away from it in 12. The average amount of deviation from the perpendicular was 7 degrees with a range of from 2 to 10 degrees. The deviation was equally toward the right or left of the field.

The vertical axes of the blind spots for both eyes deviated from the perpendicular in parallel directions for 30 observers; in equal amounts for 9 of those examined, in unequal amounts for 21.

The average amount of deviation for both eyes was 6.75 degrees with a variation in the amount ranging from 1 to 10 degrees. The deviation of one eye in excess of this averaged 5 degrees with a range of 1 to 19 degrees difference in the latter group, of which 12 showed a greater deviation of one axis away from the vertical meridian passing through the center of the field, that is, with the axes of the two blind spots sloping in the same direction in the field but slightly diverging; and 9, a greater deviation of one axis toward the vertical meridian, that is, with the axes sloping in the same general direction but slightly converging.

The vertical axes of the two blind spots converged toward the vertical meridian passing through the center of the field in 10 instances, and diverged in 31. Of these 5 converged and 10 diverged in equal amount for each eye. For the remaining observers the axis of one blind spot showed a greater amount of deviation than did that of the other. The amount of convergence for both eyes averaged about 5 degrees with variations of from 1 to 10 degrees and a further deviation of one of the axes of 4 to 10 degrees. The average amount of divergence for the two eyes was 7 degrees with a range of from 2 to 25 degrees. There was a further divergence of one axis of less than twice the deviation for both eyes for 8 observers and from 2 to 6 times this amount for 13 observers.

For the 100 persons examined, therefore, the vertical axis of the blind spot was perpendicular in both eyes in but 10 per cent of the cases; in one eye only, in 19 per cent more of the cases. Thirty per cent showed both axes deviating from the perpendicular to right or left in the same direction, 10 per cent converging toward and 31 per cent diverging from the vertical meridian passing through the center of the field. Of the 49 cases having approximately parallel axes or a vertical axis for one of the blind spots, 16 showed a tendency for these axes to converge, and 24 to diverge from each other. The amount of deviation from the perpendicular was slight, averaging about 6 degrees and seldom exceeding 10 or 15 degrees. In one or two cases deviations as great as 25 or 30 degrees were found.

11. The average size of the blind spot was found to increase somewhat with age. The average size of 88 blind spots for persons 25 years of age or under was 10.8 sq. cm.; that of 98 blind spots for persons 26 to 55 years of age, 11.6 sq. cm. The difference of 0.8 sq. cm. is not significant for diagnosis, however, when compared to the mean deviation from person to person and from eye to eye of 1.6 sq. cm. A comparison of the distribution curves for these two groups shows that the greatest number of cases in both groups fall between 10 and 11.4 sq. cm. In the younger group, however, 39 per cent of the blind spot areas are smaller than the central value; 28 per cent larger. In the older group, on the other hand, only 17 per cent of the areas are smaller than the central value while 48 per cent are larger. That is, the distribution curve for the group under 25 years of age is skewed toward the smaller values; for those over 25 years of age, toward the larger values.

12. There is an observable decrease in the average size of the blind spot as a result of practice. For 100 of the eyes included in the study the practice effect was determined in connection with another problem. The blind spot was

obtained in each study under the same intensity of illumination with the same size test object, and at the same distance from the screen. The control of fixation in the second study was slightly less rigid, however, than that for which the norms were obtained. The practice consisted of 16 determinations of the outline of the blind spot with various colored stimuli and at different distances. The average size of the form blind spot at 33 cm. distance from the eye was then redetermined and found to be 9.2 sq. cm. or 2.2 sq. cm. smaller than that previously obtained without practice. The mean deviation from eye to eye and person to person was the same for each determination.

13. The invalidity of averages based on a small number of cases is clearly brought out by a comparison of the average of 11.4 sq. cm. with that of 8.43 sq. cm. previously published by the writer, Ferree and Rand, as the average for 10 eyes. The observers were all under 25 years of age and highly trained in the technique of the test as compared to the wide range of ages and unpracticed observers of the present study. As 6 of the eyes examined formerly were re-examined in the present study, however, and showed the same average size of blind spot as that previously obtained, the differences in result of the two series can scarcely be considered as due to change in technique. Moreover, a survey of the results for the 200 eyes examined shows the possibility of selecting additional groups of 6 to 10 consecutive blind spots giving an average no greater than that previously obtained. In view of this, it seems not improbable that at least some of the discrepancies in results obtained in earlier studies of the normal blind spot may be due to the small number of eyes examined.

14. The distribution of cases shown in Fig. 1 is similar in type to that frequently obtained when a large number of measurements have been made of some physiologic or psychologic characteristic. That is, the greater number of cases cluster about a central or median value, fewer and fewer occurring as this value is increased or decreased. A much larger number of cases than we have presented would of course, be necessary to determine whether or not the distribution of blind spot areas in nonpathologic cases fits the normal or Gaussian curve as many other physiologic tracts have been found to do, but the approximation is possibly as close as one might expect for 200 cases.

When the distribution of blind spot areas for both pathologic and nonpathologic eyes has been determined in a sufficient number of cases to be fairly representative of the entire population, each new case can be immediately placed in its own proper group the relation of which to the distribution of either pathologic or nonpathologic cases has been established. The diagnostic serviceability of such a distribution is therefore limited only by the extent to which these two classes overlap. In most physiologic traits it is difficult to draw a sharp line of distinction between the pathologic and nonpathologic cases. Similarly, for blind spot areas a certain amount of overlapping is to be expected forming a group of borderline cases the status of which it is difficult to determine. It is this borderline group, however, which is most important for early diagnosis and for which the aid of the perimetric test is most needed. Greater refinements of the test have been constantly sought in an effort to reduce the number of cases in this group. The distribution scale is of particular value in the segregation of this group for further analysis and study.

The 200 blind spots presented here form too small a group from which to generalize. There are certain conclusions, however, which may be safely drawn from the results. For instance, an examination of the distribution graphs in Fig. 1 shows only 3 blind spots in which the area is 16 sq. cm. or over and but 14 in which it is 14.5 sq. cm. or over. In the group 25 years of age or under only 4 blind spots have an area of 14.5 sq. cm. or over. Obviously, then, a blind spot having an area of 16 sq. cm. may well be regarded with suspicion and one of 14.5 sq. cm. as doubtful, and in case of a person under 25 years of age, also as suspicious. The

skewing of the distribution curve toward the larger values for the older age group should very definitely be borne in mind when interpreting the results of a given examination. The cause of this variation with age, the possible effect of changes in refraction, for instance, as well as the presence of other factors has yet to be determined. It is evident, however, that from average values alone little knowledge is to be gained either as a basis for diagnosis or for further analysis of the factors involved in the determination of the size of the nonpathologic blind spot. Not size, per se, but the relative variations in size present the problem for diagnosis. In the present paper we have attempted to show the variations in size of the form blind spot in normal eyes under a given set of conditions. The variations in the color blind spots under the same conditions and for the same eyes will be presented at a later date.

University of Pennsylvania.

References

Aubert., Physiologie der Netzhaut, 1865, p. 256.
Bjerrum., Nordisk Ophthalmologisk Tidsskrift, 1890, v. 2, p. 141; X. Internat. Med. Kongr. 1892.
Bull, O., Studien über Lichtsinn und Farbensinn, Arch. f. Ophth., 1881, v. 27, pp. 54-154.
Eichenberger, J., Untersuchen über die Variabilität von Lage und Grösze des blinden Fleckes an 184 normalen Augen, Zeit. f. Augenh., 1921, v. 46, p. 88.
Ferree, C. E. and Rand G., An Illuminated Perimeter with Campimeter Features, Amer. J. Ophth., 1922, v. 5, pp. 455-465.
Ferree, C. E., Rand, G. and Monroe, M.M., Studies in Perimetry, I. Preliminary Work on a Diagnostic Scale for the Form Field, Amer. J. Ophth. 1926, v. 9, pp. 1-10.
Ferree, C. E., Rand, G. and Wentworth, H.A., The Influence of Brightness of Surrounding Field or Background on the Size and Shape of the Blind Spot for Color, Amer. J. Ophth., 1930, v. 13, pp. 690-698.
Ferree, C. E., Rand, G. and Wentworth, H. A.,The Blind Spot for Achromatic and Chromatic Stimuli, Amer. J. Ophth., 1925, v. 8, pp. 620-625.
Fick and DuBois-Reymond, Müller's Archiv. für Anatomie, Physiologie und wissenschaftliche Medicin, 1853, p. 405.
Gradle, H. S., The Blind Spot, Ann. Ophth., 1915, v. 24, p. 637; 1916, v. 25, pp. 740-758.
Griffin, Contributions to the Physiology of Vision, London Med. Gazette, new series, 1838, v. 2, pp. 223-232.
Hannover and Thomsen, Bidrag ti'l ojets Anatomie, København, 1832, v. 6, p. 61; Hanover, Das Auge, 1852, 72.
Helmholtz, H., Physiologische Optik, 1896, p. 253.
Incze, A. Über die richtige Bestimmung des blinden Fleckes, Arch. f. Augenh., 1928, v. 99, pp. 670-677.
Landolt, Graefe-Saemisch Handbuch d. ges. Augenh. pp. 60-61.
Listing, Wagner's Handwörterbuch, 1853, v. 4, p. 402.
Peter, L. C., Perimetric Studies of the Normal and Pathologic Blind Spot of Mariotte, Ann. Ophth., 1916, v. 25, p. 261.
Traquair, H. M. Clinical Perimetry, 1927.
Van der Hoeve, J. The Size of the Blind Spot and Its Distance from the Point of Fixation in the Emmetropic Eye, Arch. Ophth., 1912, v. 41, p. 350.
de Vincentiis, G., Il comportamento della macchia cieca nell 'occhio normale e pathologie, Ann. di ottalmol. e clin. oculist, 1922, p. 50; Zentralblatt f. d. ges. Ophth., 1923, v. 9, pp. 485-486.
Wittich, Arch. f. Ophth. 1863, v. 9, p. 7.
Young, T. Miscellaneous Works of Thomas Young, London, 1855, pp. 32-34; Philos. Trans., 1801, p. 47.

OPHTHALMOLOGY IN AVIATION MEDICINE

WILLIAM F. HOLZER, M.D.
WORCESTER, MASSACHUSETTS

The ophthalmic standards for commercial air pilots are stated and the methods of examination discussed. A high standard of visual acuity, muscle balance, color vision and width of visual fields is set up, as safe handling of planes depends much on ocular efficiency. Read before the New England Ophthalmological Society, January 20, 1931.

In this paper only civil aviation in the United States is considered. Time will not permit tracing its development since the organization of the Aeronautic Branch of the Department of Commerce in July 1926. It might, however, be of interest to mention a few pertinent facts, gleaned from the latest available information, to show to what proportions this infant project has grown in the United States.

There are 166 department employees in Washington and more than 1,000 in the field. Approximately 4,000 inquiries for aëronautic information are received monthly. On the approved list are 39 schools for instruction in flying. There are 7,532 licensed air craft, 15,168 licensed pilots and 9,006 licensed mechanics. The municipal and commercial airports number 1,470; 66 of which are located in New England, 23 in Massachusetts, 13 in Connecticut, 9 in Rhode Island, 8 in Vermont, 7 in Maine, and 6 in New Hampshire. There are 99 air transfer services in operation, which carried last year approximately 85,000 passengers, 8,000,000 pounds of mail, and 2,000,000 pounds of express. The record shows one passenger fatality for approximately every 1,400,000 miles of travel over authorized passenger airways.

We all know of the combined air and rail transcontinental routes, with their time-saving advantages, but many of us do not realize that a package can be transported between Boston and New York, from shipper to receiver, in 4 hours by Western Union Messengers and the Colonial Airways System.

The Department of Commerce makes four general classifications of pilots: 1. Transport pilots who are permitted to carry passengers and engage in any type of commercial flying. 2. Limited commercial pilots who are authorized to carry passengers in and out of the same airdrome within a radius of 10 miles and to carry mail and freight. 3. Private pilots who fly only for recreation. 4. Student pilots who are under flying instruction.

Two types of physical examination are given, one for private and student pilots, and the other for transport and limited commercial pilots. The latter is more exacting and more searching. The examinations are conducted by physicians designated by the department. There are 662 medical examiners, usually working in teams of two, one examiner, as a rule, making the eye, ear, nose and throat tests, and the other completing the general physical examination.

In the ophthalmological test attention is directed to visual acuity, accommodation, ocular muscle balance, depth perception, central color vision, visual fields, inspection and ophthalmoscopy.

Visual acuity. For transport and limited commercial pilots the minimal requirement for each eye is 20/20, uncorrected. For private and student pilots the minimal requirement for each eye is 20/50, corrected to at least 20/30.

Accommodation. Candidates must be able to read Jaeger type No. 1. at 13 inches.

Ocular muscle balance. Diplopia, as tested with a red glass, disqualifies for any class, although for the lower classes, that is, private and student pilots, diplopia in the extreme limits of the field may not disqualify. For transport and limited commercial pilots further investigation is necessary. An hyperphoria of more than 1Δ will disqualify. Adduction of 8Δ or more is satisfactory if abduction is not less than 4Δ. A minimal abduction of 4Δ is required, but more than 4Δ may be accepted provided adduction is increased in proportion.

Depth perception. This test is made

at a distance of 20 ft. with the so called Howard-Dolman apparatus. This is three sides of a box, back, bottom and front, with a window facing the examinee. The box contains one fixed rod, fastened to the bottom of the box, and another rod on a movable slide, which is manipulated by the examinee. Between the rods attached to the bottom of the box is a millimeter scale. The apparatus is set up, so that the examinee sees only the centers of the rods through the opening in the front of the box. The rods are separated, and the examinee manipulates the cords attached to the movable slide until both rods appear to be the same distance from him. Not more than 20 seconds is permitted for each trial. The average, for at least three trials, must not show an anteroposterior separation of the rods of more than 30 mm. The test is made without correcting lenses.

Central color vision. Any of the standard methods may be employed. The Jennings, Stilling and Holmgren tests are recommended.

Visual fields. For private and student pilots a rough form field is considered sufficient, while for transport and limited commercial pilots perimetric tests are made for form, blue and red, with 5 mm. test objects. Essentially normal fields are necessary to qualify.

Inspection. This includes examination of the lids, lacrimal apparatus and the anterior segment of the eye. Any pathological condition which may become worse or interfere with proper functioning of the eyes under the fatigue and exposure of flying disqualifies, as does nystagmus or strabismus.

Ophthalmoscopy. The presence of ocular disease or marked abnormalities disqualifies for all classes. Attention is directed to investigation for retinal or choroidal lesions, particularly retinitis pigmentosa. A mydriatic may be used if necessary—euphthalmin being recommended.

Generally speaking no candidate is passed who reveals a manifest hypermetropia of two diopters or more. As the pilot must observe other planes, watch the ground, read instruments and look at maps, constant changes of focus are necessary. Continued accommodative effort is apt to induce a tonic action of the ciliary muscles, as well as of the internal recti muscles, which in turn may produce errors of judgment of distance. Some observers find that in high altitude flying accommodation is very often seriously affected, making instrument reading and map study difficult. This of course is an undesirable hazard.

Ocular muscle balance and depth perception play an even greater part in accurate flying than does visual acuity. As soon as the plane takes off the pilot loses the one known definite quantity necessary in the judgment of distance to which he has always been accustomed, namely contact with the ground. All verticals are more or less foreshortened, and the size and shape of known objects are varied from those of previous experience. Shadows are viewed from a totally different aspect. The speed at which the plane travels is an important factor, for generally speaking the faster the plane travels in the air, the greater is its landing speed, and speed may be a determining factor in a borderline case.

Lack of good ocular muscle balance is the most common cause of errors of judgment in making landings. The exophoric individual tends to flatten out the plane too early, having judged the ground to be nearer than it actually is, resulting in a "bumpy" landing. The esophoric is inclined to fly into the ground, having judged the ground to be farther away than it actually is. In these cases the error is one of perception, and such flyers should not be classed with those having defective neuromuscular coordination. In other words, the pilot with defective visual judgment but relatively good power of performance must be differentiated from the pilot with relatively good visual judgment but poor power of performance.

Fatigue, whether due to excessive flying, lack of sleep or dissipation, has its deleterious effects upon good flying, to such a degree that most air transport companies check their pilots every 30

days, paying particular attention to their muscle balance.

Muscle exercises and, in rare instances, operative procedure increase the efficiency of flyers exhibiting muscular weaknesses.

Good color sense is, of course, important. The pilot must be able to recognize the airport markers, signal flags and lanterns of different colors. His colored maps must be properly interpreted. As the terrain over which he is flying must be viewed from different altitudes under varying weather conditions, he must be able to recognize railroads, rivers and highways, and differentiate between woods, marsh lands and different kinds of farm lands.

Normal visual fields are essential for obtaining the widest possible view of aircraft and other objects in the proximity of the pilot. Upon the vision in the peripheral fields the flyer must depend for judgment of the pace of his plane in relation to laterally placed stationary objects. The gray horizon, so called false horizon, at night is best perceived by the rod elements of the peripheral field.

At the present time no provision is made for the study of the light sense and adaptation, which it seems will become necessary as further developments in aviation take place, resulting in more night flying, as well as more flying in inclement weather. Acute light sense and ready adaptation are certainly essential qualifications of the pilot who must distinguish readily a dark object against another object nearly as dark, or make out colors, movements and shapes against a background which affords but little contrast. If he flys at night he must recognize the guiding landmarks, roads, rivers, woods and towns in foggy or rainy weather. He must be able to fly on dark nights, or in clear or misty moonlight, and be able to fathom the confusing effect of moonlight on clouds and earth mists, as well as reflections from roads, rivers and towns. Perhaps when some inexpensive portable apparatus is devised, study of the light sense will become part of the examination.

The question of the best type of goggle is still unsettled as no type yet devised seems satisfactory in all respects. At the present time those having a fixed bridge, instead of a ball and socket joint at the bridge, and a rubber cushion which is molded to fit the individual's face seem best. In cases where correcting lenses are used, fused or cemented segments are employed. Others have adaptor rings containing the correcting lenses, attached to the frame of the goggles. The limitation of the visual field and distortion of objects viewed through the lenses usually result in their discard, except when the pilot appears for examination. A movement is under way to have a system of inspection, and a mark of approval placed on those which attain a specified standard.

Thus it may be seen that the examination of the eyes of our civil aviators is, on the whole, reasonably satisfactory, though there are still some things to be desired.

As sources of the material for this paper, the writer is indebted to Miller's "The World in the Air"; to articles by Duane, Wilmer, Howard, Dolman and Cobb, in the "Transactions of the American Ophthalmological Society" (1919); and to papers by Cooper and Bauer in the "Journal of Aviation Medicine", March and June, 1930 as well as "Physical Standards for Airplane Pilots", and numerous issues of the "Air Commerce Bulletin", published by the United States Department of Commerce.

36 Pleasant street.

EPINEPHRIN IN PROGRESSIVE MYOPIA

1. Investigative Phase*

LEO L. MAYER, M.D.
CHICAGO

Epinephrin 1 to 1000 and 1 to 100 in three minim doses in conjunctival sac and subcutaneously is shown to have no effect on the pulse rate or blood pressure of anesthetized dogs. A careful clinical investigation of 10 patients showed similar negative systemic effect of the same doses in the conjunctival sacs. From the Ophthalmological Service and the Department of Physiology, Nelson Morris Institute of Medical Research and the Michael Reese Hospital.

In May, 1927 Wiener[1] reported the use of epinephrin and cited numerous cases to show the results obtained by using a 1 to 1000 solution locally, that is, in the conjunctival sacs of individuals who showed a tendency to an increase in their nearsightedness over short periods of time.

Having been in direct contact with many of these cases over a number of years, the hope that a physiological demonstration explaining the mechanism of the results obtained, might be evolved, has been much in my thoughts. Also, it occurred to me that if a 1 to 1000 solution of epinephrin has a certain beneficial effect, perhaps a stronger concentration would bring forth even more remarkable changes. Then too, with the advent of ephedrin, a drug closely allied to epinephrin and yet having a more prolonged action, it was felt that this new chemical compound might be more efficacious for clinical application.

This initial report concerns itself with laboratory and clinical investigations of the usually obtained 1-1000 solution and also higher concentrations of epinephrin. It was thought that before using stronger solutions in the conjunctival sac of human individuals, an investigation of the effect of these more concentrated collyria in animals was indicated. Through the efforts of Mr. H. W. Letton, of the Chicago office of Parke-Davis and Company, I obtained a supply of 1% "adrenalin chloride."

Pontius[2] said "The extract of suprarenal gland has no specific effect on corneal tissue, but reduces staphyloma of the cornea by constricting the arterioles, thus relieving the pressure in the lymph areas and causing a reduction of the intra-ocular tension whereby the cornea is permitted to resume its natural form." This statement was made in reference to the use of epinephrin in staphyloma of the cornea, and seems very bold in view of the fact that Pontius was merely theorizing and had no actual scientific investigations on which to base such a conclusion. My reason for quoting this statement is to eliminate from consideration any reaction on the cornea, per se, in this report. A further investigation on this phase of the problem will be assigned to another paper in the future.

Also, the changes in intra-ocular tension due to the use of various concentrations of epinephrin, and the effects of the higher concentrations on patients with progressive myopia will not be included in this report. The former is more pertinent in a discussion of glaucoma and the latter has not been tried in a sufficient number of cases over a long enough period of time to warrant any positive conclusions.

In the second report by Wiener[3] given last year in Detroit a series of 159 cases of myopia was discussed, and it would seem that more stress was laid on the coincident vigorous exercise than upon the effect of the local applications, although this latter was also of great importance. In my discussion of this paper[4] I told of this investigative work and mentioned two outstanding cases in my series where higher concentrations of epinephrin were used, that were indicative of the value of this type of therapy. Full report on this series is of necessity postponed.

* Part of the expenses incurred in this investigation defrayed by the Max Pam Fund.

Crisp[5] in his discussion of Wiener's original paper made the following statement: "the thing that makes epinephrin a little safer to instill in the eye than to inject subcutaneously is that the patient does not get quite so much systemic effect from it."

This report attempts to show just what is the systemic effect of instilling epinephrin in the eye.

The usual laboratory set up for obtaining blood pressure by inserting a cannula in the carotid artery of an anesthetized dog was arranged and the accompanying kymograph records show that instillation of neither 1-1000 nor 1-100 solution into both conjunctival sacs has any influence on pulse rate or blood pressure. Also, the results of subcutaneous injections of like amounts of these concentrations show no marked systemic effect. Intravenous findings were as expected. A recording of respiration time is not shown on these graphs as we had determined in previous trials that this simply complicated our experiment and added nothing to the results. Only the results in one trial are shown here as repetition simply corroborated our findings.

A tabulation was made of a clinical

Graph No. 1 (Mayer). Epinephrin 1 to 1000, three minims in each eye. Upper graph records pulse and blood pressure. Lower is time marker in all graphs.

investigation of ten individuals, all under the age of 21 years with pulse and blood pressure records at various times following the instillation of graded doses of epinephrin in the conjunctival sacs. My thanks are due the numerous members of the medical staff of the Mandel Clinic for their aid in making these determinations. The changes of the size of the pupils and the differences in intra-ocular tension were also taken and will be reported in another communication.

A perusal of the tables showed only variations within the normal added to the possibility of experimental error. On some patients controls without medication over like periods of time

Graph No. 2 (Mayer). Epinephrin 1 to 1000, three minims subcutaneously.

were ascertained and showed like alterations. Also blood pressure and pulse readings were recorded at other intervals. No readings were taken after a 10 minute interval as we felt that any absorption occurring would take place during this period. No subcutaneous or intravenous injections were attempted in patients as this was considered irrelevant. Those interested in such work should consult the papers of Cori and Buchwald[6] who have contributed much to the intravenous use of epinephrin in man.

The work of Istamanowa and Chudoroschewa[7], who have correlated the work of Platz[8] and Papilian[9] should also be considered in a study of this type. Although they used intravenous methods nevertheless it is important to note that they were able to show an increase in reticulocytes, especially of the younger forms, an increase in the number of erythrocytes, and an increase of hemoglobin with epinephrin. They are of the opinion that these increases are not due to stimulated erythropoietic function within the bone marrow, but to the effect of epinephrin on the blood-vessels within the bone marrow, stimulating in some as yet undetermined

manner the migration of young blood cells into the circulation. That such a procedure may also take place with instillation methods is a possibility; and that the effect of increased, blood elements may be a factor in ocular nutrition is certainly plausible.

The animal experiments mentioned in this paper were not done to confirm previous epinephrin experiments but enabled us to be sure of the potency of our solutions.

In all the determinations the same dosage of epinephrin, 3 minims, was used, whether given as instillation, subcutaneous injection or intravenously.

Conclusions

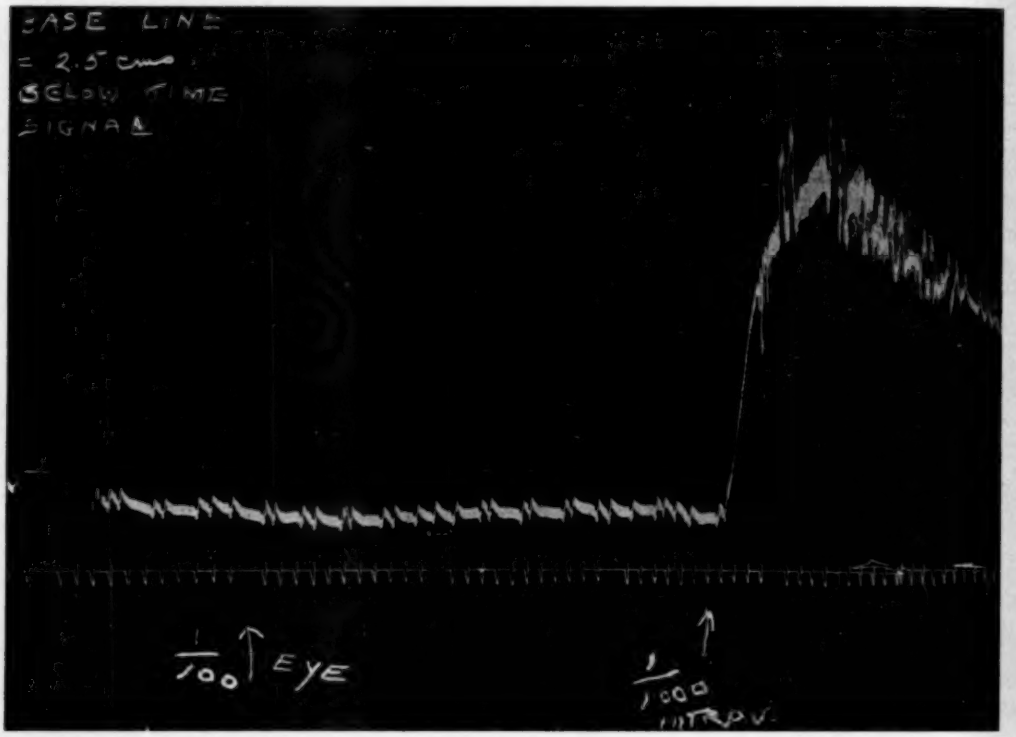
1. Epinephrin 1-1000 solution instilled as eye drops has no effect on the general blood pressure or pulse rate.

2. Epinephrin in higher concentrations up to 1-100 has no effect on the general blood pressure or pulse rate when used as eye drops.

3. It is felt that the use of epinephrin 1-100 instilled in the conjunctival sac of young people with myopia is a harmless procedure.

I am indebted to Dr. Samuel Soskin for laboratory facilities and assistance in various aspects of this work.

104 South Michigan avenue.



Graph No. 3 (Mayer). Epinephrin 1 to 100, in eye and epinephrin 1 to 1000, intravenously.

References

[1] Wiener, M. J.A.M.A., 1927, v. 89, p. 594.
[2] Pontius. Annals of Ophth., 1920, v. 21, p. 755.
[3] Wiener, M. Trans. Sect. on Ophth., A.M.A., 1930.
[4] Ibid.
[5] Crisp. J.A.M.A., 1927, v. 89, p. 596.
[6] Cori and Buchwald. Am. J. Physiol., 1930, v. 95, p. 71.
[7] Istamanowa and Chudoroschewa. Zeit. f.d. ges. exper. Med., 1930, v. 71, p. 212.
[8] Platz. Ztschr. f.d.ges. exper. Med., 1922, v. 30.
[9] Papilian. Virchows Arch., 1927, p. 264.

ANNULAR SCLERITIS

Report of a case

CHARLES W. TOOKER, M.D.
SAINT LOUIS

Annular scleritis is identified with the brawny infiltration of the sclera described by Schlodtmann in 1897. The typical form occurs usually after the age of sixty. A gelatinous infiltration gradually surrounds the cornea, accompanied by sclerosing keratitis. The disease is often bilateral and leads to blindness. The case here described was unilateral and apparently destined to recover under antiluetic treatment. Read before the St. Louis Ophthalmic Society, February 27, 1931.

There are no hard and fast lines pathologically between the deeper forms of conjunctivitis, episcleritis, and scleritis according to J. H. Parsons[1]. Clinically also the differentiation between the various types of episcleritis and scleritis is rather difficult. The severe cases have been grouped by German authors under the name "Brawny infiltration of the sclera." Schlodtmann[2] in 1897 was the first to report, from Fuchs' clinic, three cases of scleritis which he named "Sulzige Infiltration der Conjunctiva and Sklera." One of these he studied only after enucleation and one he followed clinically. This form of scleritis is usually so severe that vision is lost and the eye removed because of pain, consequently several microscopical examinations have been made and recorded. Clinically the appearance and progress of the scleritis are sufficiently characteristic to regard it as a disease entity.

Brawny scleritis (or annular scleritis) usually affects both eyes; occurs almost always in people of advanced age, and is slow in progressing. The infiltration gradually surrounds the cornea and may extend posteriorly to the equator. The swelling may overlap the cornea, and usually a peripheral sclerosing keratitis accompanies the scleral infiltration. The infiltration is gelatinous, succulent, brownish red and may be traversed by large vessels. Usually an anterior uveitis accompanies the scleral disease but opinions differ as to the primary or secondary relationship of the uveitis and scleritis. Schlodtmann and subsequent writers are not agreed as to the etiology. Verhoeff[3] in 1913, reviewed the subject, reported a case with microscopical examination of the eye, and added a bibliography. The conclusions to his paper are so clear and comprehensive that it seems pertinent to quote them.

"Brawny scleritis is a distinct type of scleritis differing essentially in both its clincial and histological aspects from anterior nodular scleritis. Clinically it is characterized by the advanced age at which it occurs (usually over 60) its insidious onset and extremely chronic course, and the diffuse congestion and thickening of the sclera and episclera without the formation of definite nodules... The infiltrated recti tendons however, may simulate scleritic nodules. Ultimately the anterior part of the sclera becomes involved around its entire circumference, and the process also invades the cornea. Evidences of intraocular involvement, such as vitreous opacities and iritis, occur late and are relatively slight. Pain likewise does not occur until late. The intraocular pressure is normal or elevated. Both eyes are usually affected although not always simultaneously, and the disease usually leads to blindness, in one eye at least. Histologically, in the early stages, there is found a diffuse plasma cell infiltration of the sclera and episclera. Later, or possibly at the same time, the sclera becomes pervaded by granulation tissue, the plasma cell infiltration increases and involves by continuity, first the anterior part of the choroid and ciliary body, and finally the whole uvea. Necrosis of the granulation tissue takes place showing itself as areas of purulent infiltration containing numerous endothelial phagocytes filled with fat, or as well-marked caseation. Giant cells may or may not be numerous. Peri- and end-arteritis are marked in the affected

tissues. Even in the advanced stages when there is intense infiltration of the ciliary body there is no formation of cyclitic membrane. Separation of the retina ultimately occurs due to exudation of serum from the choroid."

Since this publication very few cases have been reported judging from a fairly careful review of the available literature. Derby[4] in 1916 reported a case of "massive granuloma of the sclera" involving chiefly the posterior part of the eye. He stated that until 1915 only 16 cases had been examined microscopically. He believes that the pathological process "is a slow invasion of the sclera by granulation tissue extremely rich in plasma cells associated with an abundant new formation of dense fibrous tissue; within the new tissue are a few small areas of purulent infiltration and caseation."

No other cases of brawny scleritis have been reported although it is of course probable that many cases have been observed and not reported. Anterior nodular scleritis of probable tuberculous origin, as emphasized by Verhoeff[5] in 1907, has been reported by Benedict[6] in 1924, by Roy[7] in 1929 and by others. Evidently this is the more frequent form of chronic scleral infiltration. As in various other diseases typical forms may be the exception and we may designate as annular scleritis those cases which correspond fairly well to the type and certainly resemble the typical form of the disease more than they resemble other scleral diseases. The following case history lacks only a few of the characteristic features of the type of brawny scleritis described by Schlodtmann. Fortunately for the patient the eye has been retained and is useful although for purposes of scientific investigation it might be regretted that histological examination had not been possible.

Case History: T. E. H., aged 40 years, male, married, white, consulted me October 4, 1930, because of an inflamed left eye. He stated that eight days previously some dust got in his eye and that he had been attended by a physician. In 1924, I attended him for a catarrhal conjunctivitis and at that time his vision, ocular muscle balance and eye grounds were normal. On examination October 4, 1930, I found a severe iridocyclitis of the left eye. There was marked ciliary injection, a cloudy iris, a small pupil, and many precipitates on the corneal endothelium. His previous health had been excellent and with the exception of some bad teeth no history of focal infection nor of constitutional disease was obtained. Atropin and hot applications failed to enlarge the pupil adequately and many posterior synechiae were present. An x-ray examination of his teeth showed five with periapical infection and these teeth were extracted. His blood Wassermann was negative but the Kahn precipitation test was two plus. A rhinological examination did not indicate any very definite infection about the nasal sinuses, but a possible infection of the tonsils. During the following two weeks he took bichloride of mercury internally and local treatment to the eye was continued. The pupil remained rather small and irregular.

On October 11th one drop of 1% atropin was injected under the conjunctiva in the inferior nasal quadrant. The pupil became larger after a few days but on October 25th, two weeks after the subconjunctival injection, a reddish induration was noted at the site of the injection. The overlying conjunctiva was edematous and several adjacent conjunctival vessel were enlarged. The pain caused by the iridocyclitis soon subsided and during the subsequent course of the disease the eye was fairly comfortable. During the next two weeks the scleral induration extended laterally both below and above the cornea and changed in appearance. At no time did it resemble an acute inflammatory process. The infiltration on the nasal side and above, was somewhat nodular over the insertion of the internal rectus muscle. Below the cornea it extended temporally in a long sausage shaped roll to terminate near the insertion of the external rectus in a rather larger bifurcated mass. The middle third of the sclera above the cornea was not affected. The incompletely annular infiltration did not overlap the

cornea but extended posteriorly to an undetermined distance. The overlying conjunctiva was not closely adherent to it but conjunctival vessels over and near it were decidedly engorged. The color of the infiltration was a brownish red and it looked waxy, especially after pressure on the conjunctiva over it. Corneal involvement occurred later but was not pronounced. The intraocular tension was not increased.

On Nov. 5th, his physician, after another general examination, discovered some pus in his prostate gland and gave him the usual treatment. He obtained the history of a gonorrhoeal infection twenty years ago. After each prostatic massage the iridocyclitis became more active for a few days but by this time the pupil was fairly large and the ocular exacerbations promptly subsided. He developed an atropin angioneurotic oedema and scopolamine was used instead of atropin. Iodides and bichloride were used internally. On Nov. 14th his iridocyclitis seemed more severe and his rhinologist removed his tonsils. His iridocyclitis improved but the scleral induration was unchanged. There was not much ciliary injection and the eye ground was about normal.

On Dec. 6, 1930, his blood Wassermann was negative and the Kline reaction ++. On Dec. 11th to 14th intradermal tuberculin tests with 1-1000 mgm., 1-100 mgm., and 1-10 mgm. were negative. Dr. H. J. Howard examined the patient in consultation and suggested a provocative injection of neoarsphenamin intravenously. The scleritis was improving, perhaps because of the internal medication, and his iridocyclitis was subsiding. On Dec. 30th, a marginal keratitis developed at the temporal limbus but readily yielded to treatment. On Jan. 5, 1931, .45 gm. of neoarsphenamin was given intravenously and the blood examination two weeks later was negative for the Wassermann reaction but three plus for the Kahn test. On Jan. 19, .9 gm. neoarsphenamin was given intravenously. On Jan. 31, 1931, the scleral infiltration seemed smaller and the iridocyclitis had subsided leaving only a few iridic tags on the anterior lens capsule. Vision, O.D. was 20/20; vision O.S. was 20/30. Scopolamine gr. ½ to the ounce has been used 3 times a day locally for several weeks. The course of the disease will determine the nature of the subsequent treatment but probably intravenous injections of neoarsphenamin and of iodides will be needed. I hope to report the further course in a subsequent communication.

Comment. Clinically this case differs in several particulars from the type of brawny scleritis described by Schlodtmann. The infiltration was not completely annular, it did not overlap the cornea and a sclerosing keratitis did not occur. One eye only was involved and the patient was much younger than those whose case histories are reported in the literature. In other respects clinically the case corresponds to the type. A histological examination is lacking to identify the pathological process. The scleritis seems to be related to the trauma of the subconjunctival injection of atropin. The iridocyclitis preceded the scleritis and may be a separate manifestation of a general infection. Because of the probability of a traumatic excitation of the scleritis and because of the favorable course of the disease in this case, I did not excise a portion of the infiltration for microscopical study.

Although the etiology of these cases is uncertain it is probable in this case that syphilis is the cause of the iridocyclitis and of the scleritis and that the two diseases here present are related to the general luetic infection and not closely related to each other. No evidence of tuberculosis was obtained and although the infiltration was somewhat thickened over the insertions of the external and internal recti muscles, the bulk of the scleritis was brawny and almost annular. The focal infections about the teeth, tonsils and prostate probably intensified the scleritis because of the absorption of toxic products but were not truly causative. The improvement under antiluetic treatment may be only coincidental, but the history of other cases of this type indicates that such eyes are almost always lost, and if the scleritis in this eye

is resolved and the eye becomes normal it is reasonable to suppose that this scleritis was of luetic origin. The various blood examinations were of course not conclusive evidence of syphilis. The trauma of the subconjunctival injection of atropin may be considered an inciting cause of the scleritis for it has often been recorded that a minor trauma such as a foreign particle embedded in the cornea has provoked an interstitial keratitis in a luetic patient.

Conclusions. The case history here presented is one of annular or brawny scleritis but is somewhat atypical. Without a histological examination of the eye, proof of this statement is lacking. In this case syphilis is the probable cause of the disease.

1006 Carleton building.

References

[1] Parsons, J. H. Annular Scleritis. Ophth. Review 1902, v. 21, p. 181.
[2] Schlodtmann, W. Arch. f. Ophth. 1897, v. 43, p. 56.
[3] Verhoeff, F. H. Brawny Scleritis. The Ophthalmoscope. Jan. 1913.
[4] Derby, G. S. Massive Granuloma of the Sclera (Brawny Scleritis) Pathological Examination by Dr. F. H. Verhoeff. Trans. Amer. Ophth. Soc. 1916, v. 16. p. 111.
[5] Verhoeff, F. H. Tuberculous Scleritis. Boston Med. & Surg. Jour. 1907, p. 317.
[6] Benedict, W. L. Etiology and treatment of Scleritis and Episcleritis. Trans. Amer. Acad. Ophth. & Otolaryng. 1924, p. 211.
[7] Roy, Dunbar. Report of a case of Nodular Scleritis with Observations as to etiology and pathology of this disease. Trans. Amer. Ophth. Soc. 1929, v. 27, p. 96.

AN IMPROVED DOUBLE ROTARY PRISM

WENDELL L. HUGHES, M.D.
NEW YORK

An attachment for the double rotary prism by which it can be manipulated with one hand is described. Some of the tests to be made with the instrument are discussed.

The usual double rotary prism as a hand instrument has been in use for some time and, while theoretically a very useful instrument, in practise it is awkward (unless it is held on a phorometer). If one wishes to use it individually both hands are necessary, one to rotate the handle and the other to keep the prism from turning with the handle.

I have devised a simple attachment that will fit on the ordinary double rotary prism* to permit of its use with greater facility than has heretofore been possible. An enveloping tube passes over the short handle of the prism and is attached to the prism frame. At one place the sides of the tube are cut out, and fitted around a knurled knob attached to the inner shank which controls the rotation of the prisms. An extension fits into the end of the outside fixed tube to allow the little finger to hold the prism and keep it from turning around as the inner shank is rotated by the thumb and forefinger on the knurled knob. We have then a double rotary prism (I find two 15° prisms most useful) that may be used with one hand.

Fig. 1 (Hughes). Showing handle of prism taken apart and small case in which it can be carried in a vest pocket.

Fig. 2 (Hughes). Showing prism handle connected and ready for use.

I would commend this complete instrument to those who are at present using the individual prisms, or a prism rack, as being much more compact and saving much time, thereby rendering examinations in which prisms are used, much less laborious.

Those who are using the regular double rotary prism, other than on a

* The hand instrument as made by the American Optical Co.

phorometer, will find that with this attachment much time may be saved, and that the bulk of the instrument is not increased to any appreciable extent.

This instrument enables one to have available immediately any strength, from 0 to 30 prism diopters, with base in any direction, under control with one hand while the other hand may be kept free for holding the object light for testing at near or far range or a cover for one or the other eye, colored filter, etc.

The base may be applied in any direction by turning the handle through 90 degrees as the prisms may be rotated to have base either toward or away from the handle without changing its direction. By putting the handle in a vertical position the prism may be placed either base up or base down.

In testing for heterophoria with the Maddox rod in the trial frame, the least prism that will bring the streak of light through the object light, and the strength that takes the line just past the light may be determined very quickly, thus taking into account the effect of the fusion sense which makes the result of the Maddox rod test unreliable at times.

Prism convergence can be measured by gradually increasing the strength of the prism base out, until binocular vision is broken up, or by decreasing an insurmountable prism base out until the diplopia so created may be overcome, the latter usually being somewhat less than the former.

Similarly the two values for prism divergence may be simply and expeditiously ascertained.

Limits of sursumvergence and deorsumvergence may likewise be quickly measured.

These tests may be as easily and as accurately measured with the correcting lens (which one has ascertained for the patient), in the trial frame, as with a large phorometer. I have found this to be just as convenient to use and more accurate than the latter because of the fact that a slight tilt of the head does not alter the relative position of each lens to the corresponding eye. Especially is this important in vertical errors and small horizontal errors with lenses of any considerable strength where decentering of the lenses may give false heterophoric readings and unreliable data.

For exercising the various associated muscle functions, convergence, divergence, sursumvergence, and deorsumvergence, this instrument may very conveniently be used, by gradually increasing the strength of the prism in the desired direction (base in, down or up respectively). If more than 30 degree prism dioptre strength be required a fixed prism may be held before the other eye in addition.

I find the double rotary prism to be quite accurate, more so, when the values are over 6^{Δ} or 7^{Δ} than many ordinary individual prisms, a number which I have measured and found from $\frac{1}{2}^{\Delta}$ to 2^{Δ} errror in some of them.

In conclusion may I state that with this new attachment to the double rotary prism there is conveniently available any strength prism up to 30^{Δ} base in any direction, that can be entirely manipulated with one hand; the equivalent of a complete box of prisms from 0^{Δ} to 30^{Δ}. When folded up it fits into a small Oxford type eyeglass case which can be carried in the instrument bag or even in a small vest pocket.

20 East Fifty-third street.

THE POSSIBLE RELATION OF STRONG LIGHTS IN THE MOTION PICTURE INDUSTRY TO LENS CHANGES

LLOYD MILLS, M.D., F.A.C.S.
LOS ANGELES

Of forty-nine men, between ages twenty-four and forty-five, engaged in the motion picture industry for periods of six to fourteen years, thirty-nine had normal lenses, six had vacuolation of lenses and four had cortical cataract. Exposure to strong lights used in the industry is suggested as a causative factor.

The simultaneous appearance in my practice of a motion picture star, aged 42 years, and a lesser luminary, aged 45 years, in the same industry, each having cortical cataract, gave rise to the thought that exposure to the sharp lights of this work might have had a causative bearing.

With this in mind, the facilities of the largest of our motion picture studios were accorded me and on March 11, 1931, I examined the eyes of forty-seven men who had been engaged in this industry from six to fourteen years and whose particular work of preparing lights for the sets had exposed them to possible injury for much longer periods than actors. These men ranged in age from twenty-four to forty-five years. The great majority had worked for over ten years and all had had great experience working with arc lights, while over half had had "Kleig eyes" several times.

Thirty-nine were found normal as far as lens changes were concerned; six had vacuolation of one or both lenses; one had an incipient, cortical cataract strongly marked in the upper nasal quadrant of the left eye and one had well marked incipient, cortical cataracts in each eye. No study was made of the fundi in these cases, the inquiry merely being to test the theory of a possible relation between the long years of exposure to these intense lights and lens changes. No definite conclusions, of course, can be drawn from so small a number of cases. The high incidence of lens changes in young men working in a given industry, with the occurrence of two cases of frank cataract (aside from the original two which incited this interest) is highly significant of a possible connection between the industry and the pathology.

Fortunately, arc lights are used in making motion pictures only in miniature work. The incandescent light, which is coming into universal use, is much less intense than the arc light. If ultraviolet light has a relation to lens changes these changes should be reduced to a minimum as infrared is the outstanding feature of the incandescent light. The new Eastman high-speed films again will greatly reduce the amount of illumination needed and will further reduce the risks. If, however, as Colonel Smith has somewhere suggested, the infrared is the factor in the production of cataract caused by exposure to brilliant light sources, the new forms of films should effectively control this tendency through the greatly reduced intensity of illumination which they require.

This brief note is made in order to attract attention to this possible relation and if possible, finally to establish the actual facts.

609 South Grand avenue.

THE RELATION OF THE BASAL METABOLIC RATE TO PROGRESSIVE AXIAL MYOPIA

Preliminary report

LOUIS BOTHMAN, M.D., F.A.C.S.
CHICAGO

The average basal metabolic rate in thirty cases of progressive axial myopia is found to be —13.9. A review of the literature on the relation of myopia to disfunction of the internal secretory glands is given. Read before the Chicago Ophthalmological Society, March 18, 1931. From the Division of Ophthalmology. The University of Chicago.

The frequency with which well-nourished children who had progressive myopia were seen caused me to reflect that perhaps there might be some connection between this condition and the hypofunction of the thyroid gland. The findings in thirty patients under twenty years of age with progressive myopia is herewith presented.

While the literature on progressive myopia is voluminous there is very little reference to the rôle of the glands of internal secretion in relation to the condition. We know that most progression in myopia occurs coincident with the beginning of function and development of these glands. To date no writer has offered more than the observation that in certain cases of myopia a disfunction of one or another of the internal secretory glands was involved.

In 1921 Argañaraz[1] pointed out that progressive myopia occurred most often between puberty and adolescence, the time in which all glands of internal secretion show their greatest developmental activity. He cites a family with myopia in which the patient had hypopituitary symptoms and infantile genitalia. One brother was acromegalic. It is known that hyperpituitarism occurs with myopic keratoconus and Amorette reported three such cases with hypothyroidism Argañaraz also calls attention to the fact that hemorrhages and retinal detachments often occur after the menopause and that pregnancy and lactation play a rôle in progressive myopia.

Ernest Clarke,[2] in his review, Genesis of Myopia, 1925, quotes from Sir Arthur Keith's[3] article in the *British Journal of Physiological Optics* of the same year. "Myopia is a disorder of early growth. It is in the sense of an overgrowth, not like gigantism, but more a condition of stretching. Connective tissue corpuscles that control fiber building of the sclerotic have been wrongly influenced. The fibroblasts produce shoddy work. This bad influence is inherited." Clark agrees that myopia is an inherited disease affecting the growth of the sclera. Hypopituitary conditions are not associated with myopia. This alone cannot be the condition inherited. He continues, "we know so little yet about the individual and associated effects of excessi or diminished activity of these glands, but I suggest one or more of thes may be the influence which is inherited and which affect the foetal and early infantile growth of the sclerotic. Supposing one or more of the endocrine glands has or have a controlling influence on the normal growth of the eye, and if inactive, lead to wrong growth, cannot this be inherited and even occur de novo and start myopia where there has been no previous history?"

Nicolata[4] (1929) thought that disturbances of the glands of internal secretion or their hormones in the circulation during development and growth of the individual might cause a dystrophy of the corneal and scleral structures. These disturbances alone might cause only low degrees of myopia, but associated with other acquired or inherited general disturbances, might cause high grade or pernicious myopia. He thought that the disfunction of the thymus was responsible. Scalinci[5] found fault with Nicolata because he thought that the

seat of the trouble in myopia was in the choroid, not the sclera.

Incze[6] in 1929 studied the refraction in the body types described by Kretschmer. In 188 myopic patients he found 16.5 percent pyknic type, 73.3 percent asthenic, and 6 percent athletic. He concludes that myopia is no disease entity but a part of an asthenic condition. The asthenic mesodermal structures, especially the elastic scleral structures, become thin, stretch, and produce myopia. This occurs through lowering of the intraocular pressure because of decreased adrenalin in the circulation of asthenic individuals. Meyer Wiener[7] in 1930, reporting his series of patients with progressive myopia treated with local applications of epinephrin, stated that he was impressed with the fact that many of his patients were overweight. Turner[8] in 1930, in his review on progressive axial myopia, commenting on the work of Argañaraz, and Wernicke,[9] said, in referring to the secretion of the thyroid, testicles, ovaries and pituitary glands, "The syndrome of the hypofunction of these glands predominated with the greatest frequency. They believed that the diminished incidence of axial myopia or the stabilization of a previously progressive myopia, after the completion of the sexual development, was due largely to the restored normal balances in the endocrine secretion. They state that they have never seen a case of axial myopia develop in senility."

As a means of measuring the function of the glands of internal secretion it was decided to use the basal metabolic rate which is used in general medicine chiefly for testing the function of the thyroid gland. We were aware of the fact that this could not be a measure of the thyroid function alone, but as it was the most reliable test known for such functions, it was employed. It is universally believed that a high plus rate means hyperthyroidism and a low rate, hyposecretion of the thyroid gland.

The weight in kilograms and the height in centimeters, as well as the age, refractive error, and basal rate are recorded in the table which follows. The age limits are from 6 to 20 years, the average being 10.5 years. The upper limit of 20 was chosen because Brown and Kronfeld's[10] survey showed that most cases of progressive myopia ceased progressing at that age. The average height was 143 cms., and average weight 49.8 kilograms. All the cases reported were free from any pathological condition in the anterior segment of the eye and all had normal chorοids and retinæ. They might all be classified as true axial myopia.

Only cases in which the myopia increased a minimum of one-half diopter in one year, both determinations being made under complete atropine cycloplegia, were considered to be progressive myopia. The exception consisted of eight patients with one atropine determination, all of whom gave histories of increasingly poor vision, and were supported by a record of the previous unaided vision. There can scarcely be any doubt as to the true progression in any of these cases. The increase in myopia varied from one-half to four and one-quarter diopters, the average being 1.22 diopters. The basal metabolic rates recorded are given by the DuBois standard. They range from +5 to —29. There was one plus rate in the series and one with a rate of 0. The average rate was—13.8 percent.

A review of the records fails to reveal a definite ratio between the percentage of minus found on the BMR report and the amount of progression of the myopia. The lowest BMR, —29 percent, was found in case 2 in which the myopia increased in the R.E. 2.25, L.E. 1.50 diopters in 2 years. In case 3 the BMR was zero, yet the myopia increased in each eye 3.50 diopters. The +5 percent rate was found in a case (No. 10) with an increase in the R.E. of 0.75, L.E. 1.00 diopter. Taking the series as a whole, in the thirty cases with an average increase of 1.22 diopters, the average BMR was —13.8 percent. This I think is significant.

In case No. 22 the patient first became myopic in 1927. She was first seen in 1924 and was then hyperopic. Her mother was myopic. Because she was overweight and sluggish and had a

Table

Case		Age	Ht.	Wt.	Date	1st Atr. or D. Vision	D.V.	Date	2d Atr.
1	F	10	145	52	3/ 2/28	R−1.75	1.2+3		
						L−2.00	1.2+3	11/ 8/30	R−4.25−0.50c15
									L−4.50−0.25c80
2	F	10½	140	49.1	12/29/28	R−2.00	0.8+1	12/ 6/30	R−4.25
						L−2.25−0.50c165	0.8+2		L−3.75−0.50c180
3	M	20	165	80.1	12/ 1/29	R−100−0.50c165	0.6−1	12/15/30	R−2.25−3.25c5
						L−1.50	0.6−1		L−3.00−200c165
4	M	11	141.5	34.3	9/20/29	R−2.75	0.8	10/20/30	R−4.25−0.50c180
						L−2.75	0.8		L−4.00−0.75c180
5	M	10	138	29.5	6/25/28	R−17.5	0.8+3	1/ 3/31	R−4.00
						L−1.25	0.8+2		L−3.25−0.25c90
6	F	12	161	54	2/ 2/30	R−3.00−0.50c180	0.8−3	2/ 9/31	R−3.25−1.25c180
						L−3.50−100c180	1.2−4		L−4.00−1.25c180
7	F	15	159	63.5	1/16/30	R−3.00−0.25c90	1.2−2	2/23/31	R−3.75
						L−2.50	1.2+2		L−3.00
8	F	7	137	26.4	12/18/29	R−1.75−0.25c30	1.5−3	2/21/31	R−2.25−0.50c30
						L−1.75	1.2−1		L−2.25−0.75c180
9	F	15	159	63.5	2/ 6/30	R−3.00−0.25c90	1.2−1	2/23/31	R−3.75
						L−2.50	1.2+3		L−3.00
10	M	15	162	46.2	2/ 3/30	R−1.75−0.75c180	0.8+1	2/10/31	R−2.75−0.50c180
						L−2.00−100×165	1.0+4		L−3.25−100c165
11	M	11	152.5	38.1	10/ 2/29	R−0.50	1.5+2	6/ 3/30	R−1.25−0.25c180
						L−100	1.5+4		L−1.75
12	F	10	151	40	11/13/30	R−1.75−0.25c30	1.5	11/13/31	R−2.50
						L−0.75−0.25c30	1.5		L−0.75−0.25c90
13	F	12	149	47.7	12/29/29	R−0.87	1.0	1/ 2/31	R−1.75−0.50c180
						L−1.75	1.0−4		L−2.25−100c180
14	F	14	156	64.5	9/13/29	R−3.00−0.75c180	1.0−2	9/20/30	R−3.75−0.50c180
						L−3.00−0.75c180	1.0−4		L−3.50−100c180
15	F	14	140	69	2/ 9/30	R−0.75	0.8	11/19/30	R−2.50
						L−1.00	0.8+3		L−2.50−0.25c90
16	F	8	136	43	8/26/29	R−0.50−0.25c105	1.2−4	4/18/30	R−0.75−0.50c5
						L−1.25	1.0+4		L−1.75
17	F	14	161	60.5	4/18/27	R+100c85	1.2−4	2/ 3/31	R−0.50−1.25c15
						L−2.50−0.75c170	1.2−4		L−5.25−100c175
18	M	11	141	45	8/29/28	R−2.75−0.25c80	1.5−2	9/ 2/30	R−3.75−0.50c90
						L−2.00	1.5−2		L−300−075c90
19	F	6	130	37	7/19/27	R−0.50+2.75c95	1.0−3	9/30/30	R−1.75−1.50c180
						L+0.50+2.00c70	1.0+2		L−1.75−2.00c150
20	F	9	148	60	10/ 2/29	R−0.50−0.50c15	1.5−1	10/20/30	R−1.25−0.50c10
						L−0.25c165	1.5−3		L−1.25−0.25c170
21	F	12	157	55	9/20/30	R−0.25	1.5	9/26/30	R−125
						L−1.50	1.5−4		L−2.50
22	F	15	145	69	7/11/27	R−1.00	1.5−2	9/21/28	R−4.00
						L−1.00	1.5		L−3.00
23	M	8	88	26.5	9/10/30	R	1.2	3/ 8/31	R−0.75
						L	1.2		L−1.25
24	M	13	153	44	10/23/30	R	1.2−1		R−0.25c180
						L (squint)	P of L	11/ 1/30	L−0.25c180
25	F	13	159	60.6	10/ 3/29	R	1.2−2	10/20/30	R−1.25−0.25c90
						L	1.0+3		L−1.00−0.25c90
26	M	20	163	69.9	6/ 6/28	R	1.0	10/18/30	R−1.75
						L	1.2		L−1.75
27	F	20	151	58.1	6/ 7/29	R	1.0	8/27/30	R−0.50−0.25c180
						L	1.2		L−0.25−0.25c180
28	F	18	163	89.6	11/11/29	R	1.0	1/10/31	R−2.25−0.50c180
						L	1.0		L−1.25−0.50c180
29	M	7	128.5	25.7	7/20/30	R	1.0−4	2/ 7/31	R−1.50−0.50c90
						L	0.8		L−1.75−0.50c90
30	F	17	151.5	43.8	1/ 5/28	R	1.2	10/ 6/30	R−0.75−0.50c75
						L	1.2		L−1.25−0.25c90
A	F	17	131	55	1/ 6/28	R−0.75	1.2	6/16/30	R−0.75
						L−0.75−0.25c180	1.2+3		L−0.75−0.25c180
B	M	13½	145	60	10/22/28	R−2.50−125c150	1.5−4	4/30/30	R−3.25−1.25c165
						L−3.00−1.00c180	1.2+3		L−3.75−1.00c170
C	M	18	165	54	8/ 7/18	R−1.50−2.00c15	1.5−4	7/16/22	R−4.00−3.00c5
						L−2.00−2.50c5	1.5−1		L−3.75−3.50c10

basal rate of —17 percent, she was put on thyroid extract in 1928 when her refractive error was R.E. —4.00, L.E. —3.00 diopters. She was then 15 years of age. On March 28, 1930, her myopia had not increased and her visual acuity was 1.5 in each eye.

Patient No. 16 is a sister of patient No. 22. When her myopia was found to be progressive on April 19, 1930, her BMR was —9 per cent and she was put on thyroid extract. To date (eleven months) there has been no further increase in the myopia.

In the column "Rx" the "+" indicates the patients who are taking thy-

Table

D.V.	Date	Time of Prog. in yrs.	Total Inc. Diop.			B.M.R.		
			R	L	Date	in %	Rx	Comments
1.2−1	11/ 8/30	2	3.00	2.75	11 /8/30	−21	+	My. increased R ¼D L 1D in 9 mos. during '28
1.2+1								
1.2−4	12/15/30	2	2.25	1.50	12/15/30	−29	+	Increased R1D & L ¼D in '29
1.2−4								
0.8−3	12/16/30	1	4.00	3.50	12/16/30	0	−	No Rx
0.8−3								
1.2+1	10/13/30	1	2.00	2.00	10/13/30	−26	+	No change DV 3/4/31
1.5−3								
1.0−4	2/ 6/31	2	2.25	2.25	2/ 6/31	−10	+	Rx gr. 2 daily 1 mo.
1.0−4								
1.5−1	2/11/31	1	1.00	0.75	2/11/31	−23	+	Rx gr. 2 daily 1 mo.
1.5+1								
1.5−1	2/16/31	1	0.50	0.50	2/16/31	− 7	+	Rx gr. 2 daily 1 mo.
1.2+2								
1.5−2	2/24/31	2	0.75	1.25	2/24/31	−16	+	Rx gr. 2 daily 1 mo.
1.5								
1.5−1	2/14/31	1	0.50	0.50	2/14/31	− 7	+	Rx gr. 2 daily 1 mo.
1.2+3								
1.5+2	1/17/31	1	0.75	1.25	1/17/31	+ 5	−	No Rx
1.5−3								
1.5−4	8/29/30	2/3	1.00	0.75	8/29/30	−19	+	Rx gr. 5 daily. No change DV 3/2/31
1.5−1								
1.5−2	12/29/31	1	0.50	0	12/29/30	−19	+	Rx only 6 weeks
1.5−2								
1.0	1/ 5/31	2	1.25	1.50	1/ 5/31	−22	+	Rx gr. 2 daily 2 mos.
1.0+1								
1.2+1	11/ 3/30	1	0.50	1.00	11/ 3/30	−24	−	
1.2+3								
1.5	12/29/30	1	1.75	1.75	12/29/30	−25	+	Rx gr. 5 daily. DV unchanged 2/2/31
1.5−2								
1.2−3	3/ 5/30	2/3	0.50	0.50	3/ 5/30	− 9	+	Sis. and mo. myopic. Rx gr. 2 daily
1.2−2								DV same 2/8/31
1.5−4	2/21/31	4	2.75	3.00	2/21/31	−15	+	Rx gr. 2 daily
1.5−4								
1.5−2	9/16/30	2	1.25	1.75	9/16/30	− 2	−	No Rx. No change DV 1/20/31
1.5−1								
1.2−3	12/11/30	3	5.50	6.25	12/11/30	− 8	+	Became myopic 6/19/28. Rx gr. 2
1.2+2								daily. DV same 1/20/31
1.5−3	11/20/30	1	0.75	1.25	11/20/30	− 7	+	Rx gr. 2 daily. DV unchanged
1.5−4								
1.5	11/ 6/30	1	1.00	1.00	11/ 6/30	−13	+	Rx gr. 2 daily. DV unchanged
1.5−2								
1.5−2	11/15/28	1	3.00	2.00	11/15/28	−17	+	Rx gr. 2 daily 7/11/28 on 3/28/30
1.5−3	−17							R-4. L-3. DV 1.5−2
1.5	11/ 8/30	½	0.75	1.25	11/ 8/30	−12	+	2/7/31 No change in ref. DV un-
1.5−1	−12							changed. Rx gr. 2 for yr.
1.5−4	11/ 1/30	½	0.25	0.25	11/ 1/30	−25	+	3/1/31 no change in R DV. Rx be-
	−25							cause low BMR & hist. fam. My.
1.2−4	10/25/30	1	1.50	1.25	10/25/30	−19	+	Father myopic. DV unchanged
1.0−3	−19							2/20/31. Rx gr. 2 daily
1.5−3	12/10/30	2	1.75	1.75	12/10/30	− 4	−	
1.5−4	−4							
1.5−1	8/28/30	1	0.75	0.50	8/28/30	−12	+	No change DV 2/20/31. Rx gr. 2
1.2−4	−12							daily 5 mos.
1.5−3	1/13/31	1	2.75	1.75	1/13/31	−18	+	Rx gr. 3 2/5/31. BMR −4. In-
1.5−1	−18							crease dose to gr. 5.
0.8+2	3/ 7/31	¾	2.00	2.25	3/ 7/31	−11	+	Rx gr. 2 daily 1 mo.
0.8	−11							
1.5−2	12/ 5/30	2½	1.25	1.50	12/ 5/30	0	−	DV gradually poorer for 3 yrs.
1.5−3	0							
1.2+4	5/ 2/28				5/ 2/28	−20	+	Put on T 6/6/28 BMR +2. No in-
1.5−3	−20							crease of myopia since '28.
1.5−1	4/30/30				4/30/30	+12	−	10/27/30 no increase of myopia R
1.5−4	+12							& L DV 1.5−4
1.5−2	5/ 9/28				5/ 9/28	+25		Refraction in '28 same as in '22.
1.5−1	+25							Two bros. myopic, one hyp. Latter has exophthalmic goiter.

roid under the care of their physicians. These patients are all instructed to return every three months for visual tests, and whenever there is a decrease in sight, atropine refraction is repeated to be certain whether there is an actual increase in the myopia. Thus far none has shown an increase of as much as one-half diopter. The end results will be reported at a later date, as none of the experiments has been conducted for more than a year.

The treatment consists in taking thyroid extract grs. 1 twice daily by mouth, and repeating the BMR determination in three weeks. If the rate

is still minus, the dose is increased and the BMR determination again repeated in three weeks. In this way the minimum dosage of thyroid necessary to keep the basal rate between plus five and plus ten is determined, and this minimum dosage is administered until the next refraction under atropine, a year after the treatment was begun. I have not continued the study a sufficient length of time to know how long the thyroid extract should be used, but I see no reason to fear its use so long as the basal rate does not rise above plus ten and no toxic symptoms develop.

A word of warning about the reliability of some tests is in order. Care should be taken in the choice of the laboratory making the BMR determinations. One of my patients was sent to her family physician, who made the test and reported the rate to be +3 percent. The patient was an overweight, short girl with sluggish mentality. I had the test repeated at the laboratory of Billings Hospital, where a double check is made on all cases, and the rate was found to be —21 percent and —23 percent on successive days.

The cases with the minus sign in the Rx column are those in which coöperation for treatment could not be secured.

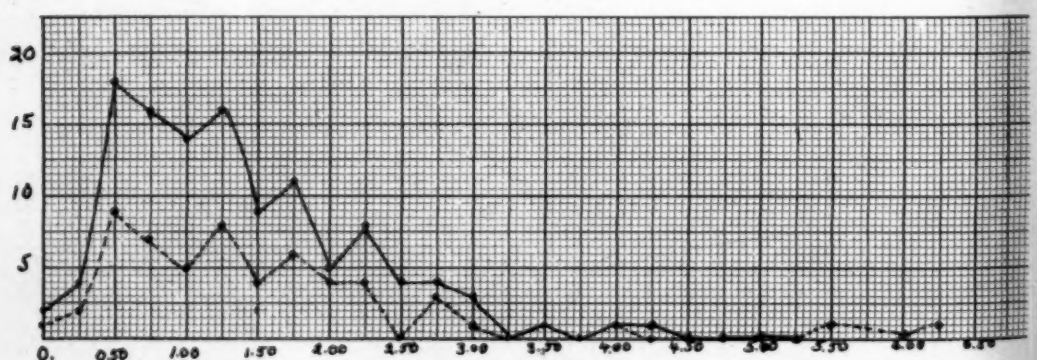
Cases A, B, and C were not included in the first series because the patients are no longer under observation. The first was a girl who developed myopia in 1928. She was much overweight, sluggish mentally, and had a BMR of —20 percent on May 2, 1928. She was given thyroid extract by her physician, from whom these facts were obtained, and on June 6, 1928, her BMR was +2 percent. She was kept on thyroid for two years and when her refraction was tested on June 6, 1930, her myopia had not increased.

Case B was of a boy who was overweight and whose myopia had ceased to progress on April 30, 1930. It has not progressed since. His basal rate on the last day was +12 percent. I have no record of the BMR during the time his myopia was progressing.

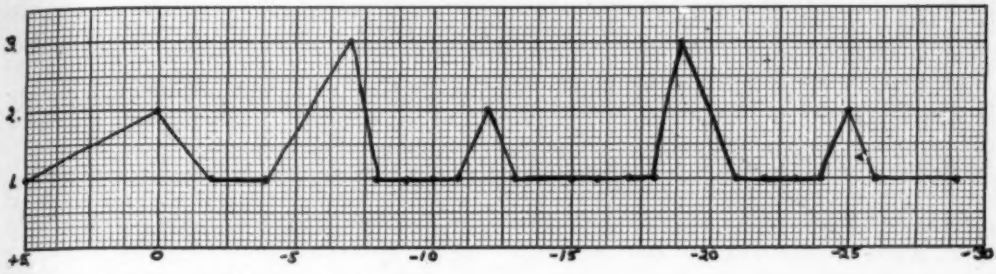
Case C is similar to Case B. The patient's parents and two brothers were myopic. His myopia progressed until 1922, since which time it has been stationary. A basal rate taken in 1928 was +25 percent. He had no goitre symptoms. The only hyperopic member of his family is a brother who had exophthalmic goitre.

A series of ten cases of patients (both sexes) ranging in age from 21 to 29 years was studied. They had basal rates from 0 to —32 percent, the average being—10 percent.

A series of fifteen cases of patients with hyperopia was studied for comparison. The sexes were mixed and ages ranged from 13 to 20. None of these patients had any general or ocular disease. The refractive errors ranged from +1.00 D. to +8.00 D. The basal rates varied from —4 percent (the only



Graph 1 (Bothman). Ordinates indicate the number of meridians.
Solid line indicates progression in diopters or fraction thereof (meridians).
Dotted line indicates progression in diopters or fraction thereof (total errors).
Average progression was 1.22 D.



Graph 2 (Bothman). BMR in a series of 30 cases.
Ordinates indicate the number of cases.
Abscissas indicate the basal metabolic rate.
Number of cases above —10%.....20.
Number of cases below —10%.....10.
Number of cases zero or below..... 3.
Average rate —13.8%.

minus case) to +18 percent. The average in this series was +8.5 percent.

The fact that some of the basal rates in the thirty cases tabulated are within normal limits, though slightly on the minus side, will undoubtedly give rise to criticism.

Thompson and Thompson,[11] in 1931, reported on a series of cases without myxedema. They state, "It is a matter of interest that most variations of the BMR from +1 percent to —10 percent fall below the zero line." In a series of 20 cases on which the BMR was run, after the patient had been confined in the hospital overnight the rate was —6 percent less than the previous rate taken in the clinic. DuBois recently stated that his standards are about 6 percent too high. With these facts in mind, the low rates of some cases in this series cannot be discounted.

The standards used at present for the BMR vary for children. A new standard is now being compiled at the University of Chicago for children under 15 years of age. It is the opinion of the investigators that the Boothly and Talbot figures, as well as those of DuBois, are too high. For this reason, too, I have included in this series only six patients under ten years of age. The average BMR of these six cases is —10.5 percent.

It must be remembered that myopic children are, as a rule, less active than hyperopic, and usually prefer to remain indoors and read. We know that inactivity leads to a lower basal rate than normal for the age. In considering this question, we must therefore ask, "Do these patients have a low rate because they are myopic and therefore less active, or are they myopic because they have a low rate and hypothyroidism"? Some patients show other signs of decreased thyroid function, such as sluggish mentality and overweight for their age. Some few patients were athletic types in which this phase of the question did not enter.

In a series of patients with hyperthyroidism (exophthalmic goitre) only 5 percent were found to be myopic and none of these had progressive myopia.

There is much to be considered along the lines of the relation of the disfunction of the glands of internal secretion and myopia. I have been unable to find any figures on the refractive errors in cretins, patients with myxedema, Addison's disease, acromegaly, hyperpituitarism, hypothyroidism or hyperthyroidism. It is my intention to gather data on all such cases in conjunction with the continuation of the present investigation. I hope to be able to follow this series of cases, along with others not tabulated, together with any new ones which may come under my observation and at a future date report the outcome of this work.

Conclusions

1. This is the first series of cases of progressive myopia in which a study of

the basal metabolic rate has been made.

2. The basal rates in this series are uniformly low.

3. Since a low basal metabolic rate indicates hypofunction of the thyroid gland, we may assume that, possibly, there may be some relation between reduced internal secretion of the thyroid gland and progressive myopia. The fact, that some cases ceased to progress after thyroid extract was taken, and that others, which were no longer progressive, had a plus basal rate, seems to substantiate this view.

4. I do not claim to have brought to light the sole cause of myopia. My intent is merely to place these facts on record so that I may interest other observers more quickly to arrive at a decision as to the merit of the assumption that hypothyroidism is a factor in causing progressive axial myopia.

122 S. Michigan boulevard.

References

[1] Argañaraz, Raul. Semaine Méd., 1921, v. 49, p. 1161.
[2] Clarke, Ernest. Genesis of Myopia. Trans. Ophth. Soc. United Kingdom, 1925, p. 373.
[3] Keith, Arthur. British Jour. of Phy. Optics, 1925, Jan.
[4] Nicolato, A. Della genesi della miopia nota prev., Archiv. Ottalm. 1929, v. 36, p. 453.
[5] Scalinci, Noë. Sui fattori biogentici delle miopia. Groin Acul. ecc. 1930, v. 7, p. 45.
[6] Incze, Anton. Über die Myopie als eine konstitutionelle Veränderung. Zeit. f. Augenh. 1929, v. 67, p. 20.
[7] Wiener, Meyer. Use of Epinephrin in Progressive Myopia. Jour. Amer. Med. Assoc., v. 89, No. 28, p. 594. Tr. Sect. on Ophth., A.M.A.1930, p. 256.
[8] Turner, H. T. Discussion of the Elements Underlying Progressive Axial Myopia. Penn. Med. Jour., 1930.
[9] Wernicke, Otto. Semaine Méd. 1921. v. 30, p. 97.
[10] Brown and Kronfeld. Refraction curve in the U. S. A. XIII Amsterdam Congress, 1929.
[11] Thompson, F. M. and W. O. Arch. Internal Med. 1930, Nov., v. 46, p. 879.
[12] Jabolinski, Walter. Eine neue Theorie über die Entstehung der Kurzsichtigkeit. Deutsch. Opt. Wochenschrift, 1927, v. 13, v. 50, p. 681.
[13] Siegrist, A. Refraktion und Akkomodation des menschlichen Auges. J. Springer, Berlin, 1925.

NICKEL SENSITIZATION FROM WHITE GOLD SPECTACLE FRAMES

A. W. McAlester, Jr., M.D., and A. W. McAlester, Third, M.D.
KANSAS CITY, MISSOURI

Three cases are reported in which different degrees of dermatitis and conjunctivitis occurred from wearing so-called white gold spectacle frames.

In June, 1931, we were consulted by a man 31 years of age, giving a history requiring corrective lenses for the relief of his headaches. He purchased a pair of white gold rimless spectacles from a well-known optical house. About three weeks later he returned, complaining of itching of the inner canthus, spreading to the inner half of his brow. A smear from the caruncle showed only mucus and pus cells. He was given an eye wash containing zinc and was told to return in several days. He complied with this but was no better. I sent him to a dermatologist, who reported he was sensitive to some material, and suggested several things, among them metal and the rotogravure of the newspaper. This man disappeared until the last of August, when he called one day stating he had lost his frames and wanted the prescription for a new glass. This time he was given a zylonite frame. His itching and erythema immediately disappeared.

The second case was of a man 46 years of age, who came for a presbyopic correction. This was in January, 1931. He purchased a pair of white gold frames. About two months later he returned with both lids covered with small papular lesions and several fissures that were moist, especially at the outer canthus. The conjunctiva had the appearance of a beginning atropin reaction. There was an erythema over the ears and at the ala of the nose. A fungus infection was immediately ruled out. He was instructed to take off his glasses and use them only when he had to see small print, which was about ten minutes a day. He was given one-half percent zinc ointment and told to use cold packs. He returned in one week, very much improved. At that time a nickel coin was strapped to his forearm and left there for thirty-six hours. He returned with a rash on his fore-

Fig. 1, Case 2 (McAlester). This picture was taken after the eruption was partly cleared. Note lesion on nose and outer angle of left eye.

Fig. 2, Case 2 (McAlester). Lesion on fore arm produced by strapping a nickel coin for thirty-six hours. Same type lesion as first noted on face.

arm identical with that he had on his face, undoubtedly nickel dematitis. It was approximately three weeks before his lids and conjunctiva were completely healed. His arm cleared up in ten days. The erythema on his nose was caused by constantly pulling on a white gold watch chain with the thumb and index-finger of the right hand, and then pulling at the nose.

The third case was of a man 54 years of age, wearing white gold frames. His complaint was, that after wearing his frames constantly for two or three days at a stretch, he developed an itching of the upper brow. This man wore his glasses only four or five hours a day, and only five or six days a week, alternating to a pair of shell frames with the total reading correction. He was suspected of being sensitive to nickel, so a coin was left on his arm for forty-eight hours, but it produced only an erythema, which was quite itchy. He was questioned about his white gold collar button, and a black spot was noted on his neck, which, however, did not itch. He changed to a pearl button and the discoloration disappeared. We found later that the original button was chromium plated and not white gold.

The cases illustrate three types of nickel sensitiveness. The first was a mild inflammation of the caruncle with an irritation of the brow. The second was that of a man, highly sensitive to nickel, with a dermatitis over the lids, ears, and nose, and a conjunctivitis. The third case was of a man, only slightly sensitive to nickel, manifesting only itching and requiring forty-eight hours to get an erythema from a nickel coin strapped to the forearm.

I could not elicit any history of other allergic reactions from cases two and three; no attempt was made in case one. Perhaps sensitiveness to metals is the cause of sore noses, where padded bridges are worn.

The following metals are in the types of frames mentioned below. I was not informed as to the percentage content of each metal.

Natural—fine gold, silver, copper, zinc.

Rose gold—fine gold, copper, nickel.

White gold—fine gold, copper, nickel, zinc.

2003 Bryant building.

OCULAR SYMPTOMATOLOGY IN ACUTE POLIOMYELITIS

HENRY M. EMMONS, M.D., F.A.C.S.
BOSTON

Ocular findings in seventy cases of this disease are reported with a summary of similar reports of the larger epidemics. Five percent of all cases in these combined reports show eye involvement. In twelve percent of the author's cases the eyes were involved. The opinion is expressed that this is more nearly the percentage to be expected. Read before the New England Ophthalmological Society, November 19, 1929.

Acute poliomyelitis is an infectious, contagious disease resulting from the growth of a filter-passing virus in the central nervous tissues, with symptoms first of a systemic infection. Exhaustive experiments made on monkeys at the Rockefeller Institute have established the fact that there is no indication that the virus multiplies in any tissue except the central nervous system and is confined principally to the tigrolytic elements of the nerves.

There is from 10 to 15 percent increase in polymorphonuclear cells of the blood.

The spinal fluid is clear, under only slight pressure, with a cell count of from 40 to 500.

The following is a report of ocular symptoms in seventy cases of infantile paralysis examined, during the last ten years, within two weeks of the onset of symptoms. The majority were studied at the Haynes Memorial Hospital for contagious diseases, following the positive diagnosis by members of the staff and by the Harvard Commission.

In this series, including many cases in the Watertown epidemic, there were twenty positive findings:

Paralysis of the internal rectus: 3 (1 with unequal pupils).

Paralysis of the external rectus: 2 (1 diplopia).

Ptosis of the upper lid: 2 (1 definite of right; slight of left).

Nystagmus: 2.

Unequal pupils: 4.

Sluggish pupils: 5.

Dilated above normal: 2.

This is a total of 29 percent. All but one of these cases were followed up and showed a return to normal within three months. Ruling out all findings except definite paralysis in order to make a comparison with the findings in other epidemics, abnormalities existed in ten percent.

The larger epidemics have been reported as follows:

Medius, in 64 cases, 9, or 14 percent, showed ocular involvement.

Wickman, Swedish epidemic of 1905, in 868 cases, 56, or 6½ percent, showed ocular involvement.

Leagard, Norwegian epidemic, in 311 cases, 6, or 2 percent, showed ocular involvement.

Zappert, Vienna epidemic of 1908, in 290 cases, 25, or 9 percent, showed ocular involvement.

Sautter, Philadelphia epidemic, in 669 cases, 19, or 3 percent, showed ocular involvement.

During the New York epidemic of 1907, in 752 cases, 44, or 6 percent, showed ocular involvement.

This makes a grand average of 5 percent. In only the first epidemic were the cases studied by an ophthalmologist. In the other epidemics the data were based largely on observations of house and medical staffs. This might well account for the differences in the percentage of positive findings. It is pretty safe to say that an average of 10 percent can be found. It is interesting to note that no mention of paralysis of ocular muscles in infantile paralysis could be found in the books on general medicine published during the last forty years. Also no objective eye symptoms are mentioned in any works on ophthalmology except in the latest edition of Fuchs's textbook where this statement is made: "Acute ophthalmoplegia occurs rarely in Wernicke's polioencephalitis superior". All later writers consider this disease not an entity but classify it as infantile paralysis.

In a review by Johns of Boston of American magazine literature on the

subject, only seven isolated cases were found: four by Posey and Swindells in the Ophthalmic Record, December, 1916; two by Sautter of Philadelphia, Annals of Ophthalmology, October, 1917; and one by Gormley, Journal of American Medical Association, February, 1921. Posey and Swindells included a case of atrophy of both optic nerves with paralysis of the left abducens; one bilateral ptosis; one paralysis of the left sixth nerve; one diplopia with complete ophthalmoplegia. Sautter's cases quoted one of bilateral external ophthalmoplegia and one of paralysis of dextro-versions; Gormley's one of optic neuritis.

There is rarity of involvement of the optic nerves in uncomplicated poliomyelitis. The first and last of these cases are open to question as there is no record of a blood-count nor of a spinal fluid cell count. According to Weisenberg all cranial nerve palsies in infantile paralysis are due to nuclear or inter-medullary lesions. Most authorities are agreed that lesions of the optic nerve are due to a complicated meningitis, Wickman claims to have seen one case of optic neuritis and also refers to a case of amaurosis with optic nerve atrophy reported by Tedeschi. Examination post-mortem in one of his cases showed on the under side of the chiasm definite round cell infiltrations of the pia. This he believes might well be extended and give rise to true optic neuritis. Posey and Swindells report a case of simple optic nerve atrophy. The resemblance of the atrophy to a simple atrophy suggests a toxic rather than a meningeal origin, according to some authors. Müller found no fundus abnormalities in the cases he studied. Uhthoff states that literature contains no mention of the optic nerve being affected in infantile paralysis. Müller, who examined some fifty cases of this disease during the acute stage without finding evidence of optic neuritis states that an optic neuritis or a choked disc speaks strongly against a Heine-Medin disease. The Rockefeller Institute reports that the eye grounds were examined in many of their cases but no abnormalities of the optic nerves were found. Examination of thirty cases of poliomyelitis at the Peabody Home for Crippled Children failed to reveal any cases showing eye ground changes.

Conclusions

1. Taking into consideration the fact that in this series of cases reported above and in the series reported by Medius, an average of 12 percent shows definite paralysis of the ocular muscles of the eye, it would seem of enough importance to general medicine to make note of the fact in all general medical literature on the subject of poliomyelitis. All works on ophthalmology should also include definite reference to the ocular palsies in poliomyelitis.

2. Due to the fact that the nuclei of the nerves which supply the ocular muscles are in close proximity to the respiratory centers, it is important that all cases of this nature should be quickly recognized as emergency cases and be immediately provided with convalescent serum.

354 Commonwealth avenue.

References

1. Tice. Practice of Medicine, v. 5.
2. Sautter, A. C. Annals of Ophth., 1917, v. 25, October, p. 54.
3. Gormley. Jour. Amer. Med. Assoc., 1925, February 21.
4. Posey and Swindells. Ophth. Rec., 1916, December, v. 25, p. 609.
5. Tedeschi, E. Paralysi Spinali Infantile Acuta Conematrofia Faccioli Ed. Atrofia del Nervo Optico Atti dell' Accademia di Scenze Medichi E Naturali in Ferrara, 1904.
6. Wickman, I. Beiträge zur Kenntnis der Heine-Medinschen Krankheit, Berlin, 1907. Wickman, I. Studien über Poliomyelitis acuta. Arb. a.d. Path. Inst. d'Univ. Helsingfors, 1905, v. 1.
7. Müller, E. Ueber die Frühstadien der spinalen Kinderlahmung München, med. Woch., 1909.
8. Peabody, F. W., Draper, G., and Dochez, A. R., A clinical study of acute Poliomyelitis. Monograph 4, 1912, June 24.
9. Wickman. Lewandowsky's Handbuch der Neurologie, v. 2, Ophth. Rec., 1916, Dec.
10. Uhthoff. Graefe-Saemisch, v. 11, 2, p. 308.

OCULAR DISEASE AND THE VERMIFORM APPENDIX

EDWARD BELLAMY GRESSER, M.D.
NEW YORK

A case of brawny scleritis and two cases of posterior uveitis are reported, which recovered so quickly following removal of acutely inflamed appendices that causative relation seemed probable. Similarly two cases of otherwise unexplained subconjunctival hemorrhage are cited. The nervous mechanism by which referred ocular phenomena might arise from irritative intestinal foci is described. From the Ophthalmological Department of New York University and Bellevue Hospital Medical College.

Low grade chronic appendicitis is usually considered a common focus of infection and numerous case reports have been utilized to illustrate this attiude.

McGuire[1] reported a case of acute uveitis dependent upon focal infection in the appendix in a girl 16 years of age. The inflammation in the appendix was chronic. Marked improvement occurred in the vision within a few days after its removal. Within 18 months the eye was evidently free from secondary changes.

According to Kritz[2], acute appendicitis is always hematogenous and never primarily caused by infection within the lumen of the appendix. Elaborating on this point it is important to recall the work of Rosenow[3] in his bacteriological studies in appendicitis, in which he stresses the specificity of certain types of bacteria for the appendix and the metastatic infecting ability of such organisms for various types of disease of the body. We find, further in this work, a preponderating percentage of recovery of the organisms from the acute cases. Similar strains of organisms had been isolated from tonsils and acutely inflamed appendices showing the predilection of that type for lymphoid structures.

Injection of such strains into animals resulted in a majority developing hemorrhagic lesions of the appendix; and strains from acute appendicitis produced lesions more marked than those from the chronic forms of these conditions. His experimental animals injected with strains of bacteria obtained from inflamed appendices suffered inflammation of muscles, joints, intestinal tract and heart. Accordingly, Rosenow considers that foci of infection contain bacteria which can reproduce these diseases in animals. These experiments suggest strongly that such foci are important factors in the production of these diseases in man.

It is surprising that with such work as a basis, clinical observations of acute appendicitis as a focus of infection have been rare. If we can accept the hematogenous infection of the appendix and hence, of other tissues, the following case reports are striking.

A young woman (D. B.) aged 26 years, presented a typical brawny scleritis of the right eye that was resistant to all usual forms of therapy. A thorough investigation failed to reveal any focus of infection, or metabolic disorders. At this time an acute inflammation of the appendix developed, necessitating operation. The day after operation the eye had become noticeably improved and at the end of another day all signs of scleritis had practically disappeared.

Paralleling this case was that of a youth (H. L.) 18 years of age, who complained of rapidly failing vision in the left eye accompanied by little pain. The history was of little help in diagnosis and the duration of the process was evidently of no more than two days. The cornea was slightly hazy and surrounded by injection. The posterior corneal surface had several punctate deposits. The aqueous was relucent and turbid. The iris was slightly muddy and its markings indefinite. The fundus could not be clearly seen due, mainly, to the turbidity of the vitreous. In short, this was a typical acute uveitis. The patient was hospitalized for investigation. The day following admission he presented symptoms of acute appendicitis.

Twelve hours after removal of the appendix he complained of severe pain

in the affected eye, which was occasioned by an increase of the intraocular tension. This complication was adequately controlled by local medication and relieved by the following day.

Recovery from the uveitis was unusually rapid in comparison with the typical course of such disease. Improvement in vision was noted on the third day after operation and continued steadily thereafter. At the end of twenty days, without any treatment, other than atropinization and compresses, no floaters were observed in the aqueous. The vitreous was free of opacities, the vision normal and a small area of healed chorio-retinitis was seen in the temporal periphery.

In a third case L. K.*, a girl, 16 years of age, complained of sudden blurring and obscuration of vision of the left eye. Her past history was negative as in the preceding cases. The ocular examination, three weeks after onset, revealed an acute uveitis and neuroretinitis. A general examination made at this time revealed a definite mild colitis. The left antrum and sphenoid having been reported as cloudy by roentgen ray, were opened. At this time abdominal symptoms became dominant and an acute pus appendix was removed. The condition of the optic nerve, retina, and cornea began to improve shortly after, and though vision had not been entirely regained, the disease process was arrested.

The foregoing cases were suggestive of correlation of acute ocular disease and acute appendicitis and stimulated a search for further examples. To date only two such have been seen. Both were of the same character and occurred in children, three and a half and five years of age, respectively, in each of whom an unexplained subconjunctival hemorrhage appeared twelve to twenty hours before the onset of acute appendicitis. These were completely absorbed in 48 hours after operation.

Unfortunately it was impossible to make a bacteriological study of the eyes in the first three cases to demonstrate whether the same organism was present in both eye and appendix. However, the celerity with which the ocular disease disappeared following appendectomy creates the impression that we were dealing with a focus of infection that presented acute manifestations rather than the chronic types to which we have been accustomed. There is also a striking similarity in the appearance of the subconjunctival hemorrhages, in the same types of appendiceal disease, this coincides with Kritz's beliefs.

Aside from the rôle of focal infection that the chronically inflammed appendix plays, the work of Eppinger and Hess shows that a state of vagotonia may be induced by this organ when irritated. Edington[4] has found that the appendix and gall bladder have both received their quota of infection from an original focus and permit of induction of vagotonia in their irritated state, through the vagus nerve network. It is common knowledge that an apical abscess provokes earache or an infected antrum causes trigeminal neuralgia. Similarly an organ innervated by a branch of the vagus may cause an hyperexcitability of the vagus as a whole. Edington also has demonstrated that this condition of vagotonia, enhanced by the use or abuse of tobacco, may cause symptoms of ulcer, or may convert an acute ulcer into a chronic one. Such an ulcer will relapse without fail if the chain consisting of focus, irritated appendix, or cholecystitis is not attended to in each link.

Eusterman[5] is not sure to what extent the appendix and gall bladder can provoke disorders, but finds the chronic appendix has been accused of producing many ills. It has been frequently shown that complete and proper removal of all tangible foci not only will cause subsidence of gastric disturbances which were without demonstrable organic basis, but such removal also affords evidence of increased healing of an otherwise refractory ulcer. The same author finds tenable the theory of ulcer by infection, contemplating as he does, the great prevalence of chronic foci harboring organisms of low grade virulence

* Personal communication from Webb W. Weeks, M.D.

which attack certain tissues of organs over long periods of time.

The mechanism by which an irritated appendix may influence the normal physiology of the eye and its appendages, is shown by the following nervous relationship.

The vagus nerve in addition to its wide distribution in the thorax and abdomen and interconnection therein with the sympathetic system has further communication in its jugular ganglion. Herein is connected the parasympathetic network through fibers from the superior cervical sympathetic ganglion and accessory nerves that pass to the plexus on the internal carotid artery. Further, the ramus auricularis from the jugular ganglion communicates with the auricular branch of the facial nerve. The facial nerve in turn is also involved in the nerve plexus on the internal carotid artery by way of the deep petrosal nerve. The sensory nerve of the eye (the trigeminal), forms part of the same internal carotid nerve plexus by a branch given off while in the cavernous sinus. A branch from the ophthalmic division of the lacrimal nerve also anastomoses with the temporal branch of the facial.

Hence, from this brief description of the inter-relationship of these nerves, it is comprehensible how remote possibilities of reflex action of both vagotonia and sympatheticotonia effects can take place. Remote effects of nervous irritability translated into symptoms and signs through such mechanisms are aptly illustrated by the case of a middle aged woman (F. G., aged 50 years) who had been troubled for several years by an annoying epiphora and vague dyspepsia. Following a cholecystectomy and an appendectomy there was a cessation of epiphora. In a patient (D. S., aged 28 years, male) complaining of frequent pain in the right eye, associated with periods of constipation and mild generalized abdominal cramps, an appendectomy relieved these complaints. An ocular examination revealed no error of refraction and normal muscle balance. In both of these cases pathological reports indicated chronic appendicitis. These cases suggest that there is a vagotonic state influenced by a chronic focus of infection. The same mechanism possibly can be invoked by an acutely inflamed and irritated focus. Though we have been unable to find cases illustrative of this point, Pearcy and Allen[6] have succeeded in demonstrating how acute gastro-intestinal upsets may become associated with visual disturbance.

Distending the stomach and intestines of man, artificially, they were able to produce hypotony of the ciliary muscles with resulting blurring of vision and eye strain. This reflex accommodative insufficiency was reproduced independently of refractive errors, extraocular muscle imbalance, or intraocular pressure.

Summary. Three cases of acute inflammatory diseases of the eye are described occurring in conjunction with acute appendicitis. In two cases the posterior segments of the eyes were involved and in one the sclera. Subconjunctival hemorrhages were seen in two cases several hours preceding an attack of acute appendicitis for which no adequate causes were found locally.

Acute ocular disease caused by a focus of infection in a chronically inflamed appendix is not discussed.

The nervous system inter-relationship that permits of referred ocular phenomena from irritative intestinal foci is described. Two cases aptly illustrate this condition.

39 Fifth avenue.

References

[1] McGuire, H. H. Uveitis dependent upon focal infection in the appendix. Arch. of Ophth., 1920, v. 49, p. 500.
[2] Kritz, R. Angina, und septische Infektion. Zeit. f. Heilk. 1907, v. 28, p. 296.
[3] Rosenow, E. C. Focal infection and elective localization of bacteria in appendicitis. Surg. Gynec. Obst., 1921, July, v. 33, p. 19.
[4] Edington, H. D. Gastro-intestinal sequelae of focal infection. Jour. Med. Assoc. South Africa, 1927, June, v. 1, p. 300.

[5] Eusterman, G. B. Certain clinical and pathologic aspects of focal infection. Ann. Clin. Med., 1924, November, v. 3, p. 384.
[6] Pearcy, J. F. and Allen, T. D. Studies on the visceral nervous system reflexes from the gastro-intestinal tract to the eye. Amer. Jour. Physiol., 1927, September, v. 82, p. 56.
[7] Bassler, A. The intestinal canal as a source of focal infection. Phys. Therapeutics, 1928, August, v. 46, p. 384.
[8] Jenkins, I. W. The colon as a primary focus of infection. Texas State Jour. of Med., 1929, July, v. 25, p. 213.
[9] Reeder, J. E. Uveitis. Jour. Iowa State Med. Soc., 1917, August.
[10] Rodda, F. C. Manifestations of focal infections in the respiratory, circulatory, urinary and gastro-intestinal systems in infancy and childhood. Minn. Med., 1924, January, v. 7, p. 27.

SENILE MACULAR CHANGES OF THE RETINA ASSOCIATED WITH FOCAL INFECTION

HUNTER W. SCARLETT, M.D.
PHILADELPHIA

A review of numerous articles is given and four cases are reported where macular degeneration was associated with foci of infection and arteriosclerosis. Some of the cases showed improved vision after removal of the foci. Read before the Section on Ophthalmology, College of Physicians, Philadelphia, April 16, 1931.

The various causes of macular changes, as well as their mechanism, are interesting and at times baffling. Such changes occur more frequently after middle life than in earlier years. When associated with a focus of infection, their etiology becomes less obscure.

Behr[1] divided macular affections according to different periods of life (a) congenital, (b) those of dentition, (c) those at the beginning and (d) end of puberty, and finally (e) those seen in old age. These last he regarded as an early indication of impending death, and considered the condition slowly progressive without leading to blindness, but with the possibility of some improvement in function.

Van der Hoeve[2] found senile changes in the macula in approximately six percent of his patients between 60 and 70 years of age, 23 percent between 70 and 80 years, and 30 percent above 80 years.

According to de Schweinitz[3] senile macular degeneration is a condition found in elderly eyes. This condition reveals areas of yellowish white spots interspersed with pigment dots and small hemorrhages or irregular areas of erosion, which may go on to atrophy or pigment heaping. Harms, by microscopic investigation, says the lesions chiefly depend upon atrophy and disappearance of the neuro-epithelial layer of the retina. The choroid is not as a rule affected.

Fuchs[4] believed that cases of senile degeneration of the macula existed in which it was impossible to determine whether or not they occurred on a basis of an anomaly of development. He examined a few cases anatomically, and found the choriocapillaris normal; so one must infer that here the degeneration started from the neuro- and pigment-epithelium and not from the choroid.

Bell[5] examined a patient with choroiditis and vitreous opacities, whose vision improved from 20/200 to 20/20 after the treatment of severe pyorrhea. A second patient, having a retinal hemorrhage, was treated with vaccines and recovered. Bell[6] believes that oral sepsis is one of the direct causes of septic gastritis, toxic neuritis, arthritis, nephritis, and arteriosclerosis, and that ocular complications may be secondary to these. McFarling[7] thinks focal infection in the teeth paves the way for the pathological results of some trivial exciting cause. He says the infection from a focus may occur in three ways: (1) by direct extension; (2) along mucous or serous membranes; (3) by

the blood and lymph channels. This latter way is most important for ocular lesions. Dewey[8] favors the lymph channels as the mode of travel of infection from the teeth to the eyes.

Van der Hoeve[9] says the eye should be the barometer as to whether an infected tooth should or should not be removed. If the eye is in real danger the tooth should be extracted, but a tooth should be considered as important as any other structure.

Haden[10] experimenting by inoculating rabbits with organisms obtained from the teeth of patients suffering from ocular diseases, found 68.2 percent developed lesions of the eye, while of those inoculated with organisms from patients without such diseases, only 14 percent showed eye lesions. Hence he considered that there was a selective affinity of bacteria for the tissues of the eye.

Longcope[11] is uncertain whether lesions distant from foci of infection are the result of metastatic microörganisms or their products, or whether the pathology is an allergic phenomenon. Little is known about the latter, when resulting from bacteria. Two substances can be isolated from bacteria, one proteid in character, the other nonproteid, and both highly soluble. The latter may produce a marked shock, after the animal has been sensitized to the first. Woods[12] says toxins do not as a rule produce eye lesions, and the allergic theory does not wholly explain the subject.

In Finlay's case,[13] macular changes were thought to be due to intestinal stasis, caused by a dilated colon, as treatment of the latter condition improved the vision, and the macular changes became almost imperceptible. Five years later a central retinochoroiditis was diagnozed as due to an apical abscess. Improvement followed treatment with autogenous vaccines. Finlay explained the macular lesions on the basis of arteriosclerosis, which developed at the slightest variation of the blood supply from the normal, the latter condition having been produced by certain toxins or poisons.

Batten[14] thinks macular changes are frequently the only evidence of disease discrepancy, and that the family history should be kept in mind in studying these conditions. In his case pyorrhoea was present.

Roll's[15] case presented an organized fibrous plaque in the macular region. Sight had been failing for several years. Cruise mentioned a similar case, with edema and stippling of the macula, followed later by a pale area, over which pigment dots could be seen. The etiology was supposed to be a tuberculous focus. From two similar cases dysentery bacilli and paratyphoid organisms were obtained. Both improved under vaccine treatment. Blair[16] also saw a case with a mass at the macula. In discussion Batten referred to a similar case following scarlet fever and middle ear disease.

Fuchs[17] described a disc-like degeneration in the macular region of aged eyes. The condition was usually bilateral. The anatomic change was a membrane between the retina and the choroid, sharply delimited toward the chorold, so that it was supposed to originate from the pigment epithelium and capillaries of the retina.

Cords[18] demonstrated seven cases with a disc-like formation in the macula. These patients had generalized arteriosclerosis and negative Wassermanns. He thought all had a common basis of origin in disturbance of nutrition due to closure of the finest capillaries.

In the last of eight cases of disc-like formation in the macula, reported by Holloway and Verhoeff[19], microscopic examination revealed a mass of connective tissue between the retina and the choroid, probably derived from the pigment epithelium. In six cases circinate exudative lesions were present. It would appear therefore that such lesions either preceded or accompanied the macular changes.

It has been quite clearly demonstrated that circinate retinitis starts from sclerosed retinal capillaries, usually with a small macular hemorrhage. The exudate follows later, taking the

form of the characteristic circinate lesion.

The type of lesion known as macular hole, may occasionally develop from a central retinochoroiditis, due to a focus of infection acting as an underlying etiological factor.

Case 1. Miss C. D. H., aged 65 years, was first seen May 29, 1924, because of black specks before her right eye. Vision O.D. was 6/6; O.S. 6/6. External examination was normal. Fundi showed marked retinal arteriosclerosis, especially of the macular vessels. She returned on June 25, 1928, complaining of a brownish yellow fog in front of her right eye, which had gradually developed during the past month. The vision of the right eye was reduced to 6/20. The left remained normal. The macula of the right eye showed a few grayish brown dots. About midway between the disc and the macula was a small area of retinochoroiditis. One year later the vitreous of the right eye was filled with opacities. At this time Dr. de Schweinitz saw the patient in consultation, at which time a small hemorrhage was apparent among dots in the macular region. Some pigment also was seen. There was a relative central scotoma for blue.

All other examinations, except the dental, were negative. A lower right molar tooth was devitalized. All the teeth appeared normal to the x-ray; however since the diminution of vision could not be accounted for, I strongly advised her to have the dead tooth removed. At the time of extraction a culture revealed hemolytic and nonhemolytic streptococcus together with staphylococcus aureus and albus. A vaccine was made and eight injections were given. Following the removal of the tooth and the injections of vaccine the vision improved from 6/20 to 6/10.

The diagnosis was senile macular degeneration of the right eye with vitreous opacities.

Case 2. Mrs. M. C., aged 70 years, when first examined complained of slightly diminished vision in both eyes. Vision was 6/10 with each eye. Vitreous of each eye contained many stringy opacities, while the discs were poorly defined. In each macular region there were a few small yellowish brown dots. The left one had a short streak of pigment running diagonally across it. The retinal vessels were markedly sclerosed, as were the vessels of the general vascular system. Fields were negative, except for a relative scotoma for colors.

The patient had badly infected tonsils, but refused to have them removed.

The diagnosis was senile macular changes in both eyes with vitreous opacities.

Case 3. Mrs. H. B. T., aged 67 years, was found to have a vision of 3/40 in each eye, with a compound hyperopic astigmatism. Her correcting glasses brought her vision to 6/6. The vitreous of each eye contained several fine stringy opacities with a large heavy one in the left eye. The disc edges were slightly blurred. The macular areas presented several yellowish white dots, with two pigment spots in the center of each macula. The retinal vessels were markedly sclerosed. The visual fields showed relative central scotomata for colors.

The patient's tonsils were badly infected as were also the left ethmoidal cells. Her left tonsil was treated with electricity and a few days after the treatment she said she noticed the vision of the left eye had become more impaired. It was reduced to 1/40, while the right eye remained 3/40.

The diagnosis was bilateral senile macular degeneration with vitreous opacities.

Case 4. Mr. S. B. R., aged 74 years, had had blurred vision for several months, with floating specks before his eyes. Acuity of vision had dropped to 6/40 in the right eye, and to 6/20 in the left eye. The cornea on each side showed a marked arcus senilis. The lens of the right eye had an incipient cataract at about "10:30 o'clock". The vitreous of both eyes showed numerous black stringy opacities. Retinal vessels were markedly sclerosed, and the macular region presented several yellowish white spots interspersed with pigment.

There was a relative scotoma for colors.

The patient had had an infected tooth extracted about one year prior to the examination, while two months before, he had had a large boggy prostate removed, but without any improvement in vision.

The diagnosis was senile macular degeneration and vitreous opacities.

Summary and discussion

Three of the four patients studied in this report were females, and all were over 65 years of age. Their macular changes were of the senile degenerative type, and advanced retinal arteriosclerosis was present in each case. This brings up the question of the rôle of such vessels in the production of macular changes.

It is easy to imagine that diseased vessels are more vulnerable to the action of bacteria or their toxins than healthy ones. If there exists an altered vascular network as an underlying factor, and then there should be poured into that vascular system the products from one or more foci of infection, it is reasonable to predicate the lesions seen in the macular changes of aged eyes. Closely allied to the changes observed under arteriosclerotic conditions, are those of circinate retinitis as mentioned by Holloway and Verhoeff.[19]

My first case was interesting from two standpoints: (1) the partial recovery of vision, and (2) the question whether the improvement in vision would have occurred after the removal of the tooth alone, or whether the autogenous vaccines were the potent factor. Both measures were undoubtedly of value.

Cases 2 and 3 had infected tonsils as associated conditions, but the macular changes did not appear to be so grave as those in case 1, although the vision in case three became more impaired after treatment of one tonsil with electricity, which probably seared over a pus pocket.

The association of an infected prostate is exemplified in case four. De Schweinitz[31] has discussed the possible relationship of senile macular degenerations to nonspecific infections of the prostate or other foci of infection, and advised the suppression of all foci during the hemorrhagic and suppurative stages of such ocular lesions.

In the case reported in this paper there was an apical abscess antedating the prostatic infection. It might well be queried whether or not the latter was possibly caused by the former.

Conclusions

1. Arteriosclerosis as a fundamental element, plus a focus of infection as a secondary activating agent, may be postulated in recording the etiology of macular changes in patients past middle age.

2. Vitreous opacities revealed the presence of a low-grade involvement of the uvea, in four cases studied.

3. A thorough survey for all possible foci of infection, should be instituted in every case showing macular changes.

4. Autogenous vaccines are of distinct value in the treatment of cases of macular changes, where a focus of infection is present.

5. The presence of a central scotoma for colors is helpful in the diagnosis of such cases as show a slight alteration of the macula.

230 South Twenty-first street.

References

[1] Behr. Heredodegeneration of the macula. Klin M. f. Augenh., v. 65, p. 465.
[2] Van der Hoeve, J. Senile degenerations of the macula and lens opacity. Amer. Jour. Ophth., v. 2, p. 749.
[3] de Schweinitz, G. E. Senile macular atrophy of the retina. Diseases of the Eye, 10th ed., p. 538.
[4] Fuchs, E. Classification of retinitis. Arch. of Ophth., v. 3, p. 393.
[5] Bell, G. H. Relations of the teeth, tonsils and intestinal toxemias to diseases of the eye. Trans. Amer. Med. Assoc., Sec. on Ophth., 1919, pp. 266-293.
[6] Bell, G. H. Teeth, tonsils and toxemia. Dental Cosmos, 1921, May.

[7] McFarling, A. C. Etiologic importance of focal infection in ophthalmic practice. Jour. Oklahoma State Med. Assoc., 1921, v. 14, pp. 54-60.
[8] Dewey, K. W. Affections of the eye from diseased teeth in relation to the lymphatic system. Arch. of Ophth., v. 49, pp. 367-380.
[9] Van der Hoeve, J. Relationship of dental sepsis to diseases of the eye. Trans. Ophth. Soc. United Kingdom, 1923, v. 43, p. 450.
[10] Haden, R. L. Elective ocular localization of bacteria from infected teeth. Arch. Intern. Med., 1923, v. 23, p. 828.
[11] Longcope, W. T. Rôle of infected teeth as seen by the internist. Amer. Jour. Ophth., 1924, v. 7, p. 470.
[12] Woods, A. Ocular lesions of dental origin. Amer. Jour. Ophth., 1924, v. 7, p. 469.
[13] Finlay, C. E. Recurring central chorioretinitis with distinct etiology. Rev. Cubana de Oft., v. 2, p. 438.
[14] Batten, R. D. Acute macular disease. Trans. Ophth. Soc. United Kingdom, 1921, v. 41, p. 411.
[15] Roll, J. W. Macular changes. Proc. Roy. Soc. Med., Sec. on Ophth., 1921, June.
[16] Blair, C. L. Macular mass. Amer. Jour. Ophth., 1922, v. 5, p. 217.
[17] Fuchs, E. Classification of retinitis. Arch. of Ophth., v. 3, p. 393.
[18] Cords. Disc-like formation in degeneration of the macula. Deut. Ophth. Gesell, 1925, p. 246.
[19] Holloway, T. B., and Verhoeff, F. H. Disc-like degeneration of the macula. Arch. of Ophth., 1929, v. 1, p. 219.

CONCERNING SIMPLE GLAUCOMA

HARRY S. GRADLE, M.D., F.A.C.S.
CHICAGO

Provocative tests for the determination of simple glaucoma in the earliest stages are described and evaluated. Illustrative charts accompany the paper. Read before the Baltimore Ophthalmological Society, January, 1931.

The indulgence of the reader is craved in expressing these more or less random personal views and observations. No attempt will be made to offer a systematic study of glaucoma nor will there be any extended discussion of the works of others nor any compilation of bibliography. Those interested in moot points will have no difficulty in finding the necessary literary references, both clinical and experimental.

In the first place, this paper deals purely and simply with simple glaucoma, also know as non-inflammatory or compensated glaucoma, or glaucoma simplex. Unless dealing with some specific point, the average author speaks of "glaucoma" and does not differentiate the type. Neither etiologically nor clinically do simple glaucoma and inflammatory or better, uncompensated glaucoma have many points in common. It is perfectly true that an old case of simple glaucoma may eventually become "uncompensated" in this sense, and it is equally true that both types have one common aspect, namely increased intra-ocular tension. I am firmly convinced that simple glaucoma and uncompensated glaucoma are two separate and distinct diseases with different etiological factors and with different clinical manifestations, that, if neglected, progress to one common end. But primarily, the two diseases are so radically different that they must not be discussed in the same breath.

As yet we know nothing of the cause of simple glaucoma. We do not even know the exciting factors as we do in uncompensated glaucoma. All that is known is that simple glaucoma appears after the age of thirty-five years, but it may appear in children; it is usually associated with low hyperopia, but it is also found in high hyperopia, low myopia, and high myopia, not to mention emmetropia; it is more common among Jews than in any other race; but so far, it has not spared any known race; and finally it is more common among sympathicotonics than vagotonics, but still the most placid of individuals are affected. In other words, simple glaucoma is supposed to appear

in a certain type of individual with a definite error of refraction, but it is equally liable to appear in any individual of any race or any age with any sort of a refractive error. There is nothing like a little variety to keep the ophthalmologist up on his mental toes.

In a frank, outspoken case of compensated glaucoma, the diagnosis presents no difficulties. But the early cases, those that lie on the border-line between normal and pathological tension, are overlooked only too frequently and it is for the detection of such cases that the ophthalmologist must be constantly alert. The classical signs of dilated pupil, decreased vision, and cupped disc with increased intra-ocular tension have not yet appeared. To the ordinary examination, the pupil responds normally; the vision may be decreased so slightly that the defect is overlooked; the cupping of the disc is still within the normal limits; and to even the trained fingers, the tension does not seem increased. What is there then about these potential glaucomas that drives the ophthalmologist to further investigation? In the first place a hint may be derived from the history given by the patient. For example, in an individual of glaucomatous age, regardless of the degree or character of ametropia present, the story of headaches appearing after a stay of twenty minutes or more in a darkened room, such as at a moving picture theatre should arouse suspicion. Again, headaches appearing during the morning hours, may demand further investigation. In some instances, a digestive upset is the precursor of a simple glaucoma, although it must be said that gastric disturbances are more frequently associated with the inflammatory or uncompensated type of glaucoma. Again, the history of glaucoma in an immediate forebear must arouse suspicion, as must a slight decrease in vision that cannot be corrected to normal.

But even without an amnestic hint, the suspicion of the ophthalmologist may be aroused by the border-line eye itself. The presence of minute dots of brown pigment on the posterior surface of the corneal endothelium in the absence of similar cells floating in the aqueous justifies further investigation. These pigment dots, which can be seen only bio-microscopically, are probably a forerunner of the Koeppe pigment distortion. A shallow anterior chamber not accounted for by a swollen lens, has to be explained. An unduly easy dilatation of the pupil in light shadow, casts a doubt upon the integrity of the ocular end of the pupillo-motor pathway. A suspicion of circum-papillary halo may be the beginning of a pathological excavation of the disc. At this point it may not be inappropriate to comment upon the lack of relationship between a physiological excavation of the disc and a glaucomatous one. In other words, no one can foretell a glaucoma by the appearance of the normal physiological excavation of the nerve-head, but in a suspicious case, a great deal can be learned by comparing the optic discs in the two eyes for seldom is the progress of a glaucoma identical in both eyes. According to Vogt, the presence of a capsular membrane on the lens (Kapselhaütchen) is pathognomonic of glaucoma, but in the two cases I have seen, there was no glaucoma nor has it developed during the course of three years.

If the suspicions of the ophthalmologist are aroused, the presence or absence of a definite glaucoma must be proved. First a thorough examination of the eyes must be made which must include accurate description of the tissues; vision, both with and without correction; peripheral visual fields; central visual fields; dark adaptation; and intraocular tension. At this point, may I digress slightly to comment upon the clinical measure of dark adaptation. The work of Derby and his associates is excellent from an experimental standpoint, but the comprehensive examination upon which his figures are based is not necessary from the clinical standpoint. The normality of light sense of a patient may be determined easily and rapidly by comparing it with that of the examiner (provided the examiner is normal). For that purpose any of the commercial

foot-candle-meters suffices. The patient and the examiner sit side by side in a dark room and after three minutes, the scale of the instrument is illuminated minimally and the reading of the patient is compared with that of the examiner. Three such comparisons at three minute intervals will suffice to discover whether the dark adaptation of the eye is normal or not. The sole information desired is whether it is reduced.

After the complete information has been obtained by this first examination, it is advisable to endeavor to reproduce the conditions under which the patient suffered the significant symptoms. If the headache or decreased vision followed attendance at a movie, put the patient in a dark room for an hour and at the end of that time, measure the intraocular tension without subjecting the eye previously to strong illumination. Less than fifty per cent of the cases of simple glaucoma will show an increase in tension of from 10 to 30 mm. Hg. or more by this maneuver. The so-called Seidel dark room test gives definite evidence when positive, but a negative outcome does not eliminate the diagnosis of glaucoma. According to Seidel, the increase in tension is due to partial blocking of the chamber angle by the thickening of the iris subsequent to pupillary dilatation. In the eye so predisposed, the pupil may dilate to a given diameter, which varies according to the individual eye; but the moment the dilatation goes beyond that threshold, the iris becomes too thick for the narrow confines of the anterior chamber and a partial blocking of the chamber angle results with consequent increased pressure. In a frank outspoken glaucoma, it is not necessary to carry out this test, but in a borderline case, it is of great value when positive.

It has been our practice to hospitalize these border-line cases for diagnosis for the purpose of obtaining a four hour curve of the intraocular tension. For the convenience of the patient as well as the hospital personnel, the tension is measured at 8 a.m., noon, 4 p.m. and 8 p.m. Anesthesia is produced by one drop of two per cent butyn. The patient always lies flat on the back during the measurement. As the resident takes all the tensions, the personal equation is eliminated as far as possible. It is realized that the highest tension usually occurs about 5 a.m. and some three hours later when we take the tension, there has been somewhat of a drop. But the relationship is constant. Unless there has been a definite diagnosis of glaucoma made, no miotics are used until at least a twenty-four hour and preferably a forty-eight hour tension curve has been plotted. During this time, the Seidel dark room test is carried out, preferably in the morning hours. During the course of the next two days we also carry out the "coffee test", the "drinking test", and the "reading test". These three tests are less frequently positive than the dark room test, and similar to that, do not deny glaucoma if negative. In other words, they are fairly conclusive when positive (in conjunction with other factors), but mean nothing if negative.

The "coffee test" consists in having the patient drink one or two cups of strong black coffee. When positive, the intraocular tension will rise from 15 to 25 mm. Hg. in from twenty to forty minutes after ingestion of the stimulant. The "drinking test" must be carried out before breakfast when the patient drinks one quart of water as rapidly as possible. The tension, measured every fifteen minutes, should rise from 8-15 mm. Hg. in less than half an hour. I personally have never seen a positive, although some German authors seem to value the test highly. The "reading test" is still less likely to be positive but is of very definite value when so. The patient is told to concentrate upon any form of reading, preferably fine print at a close range, and the tension is then measured after forty-five minutes. When positive, the increase in tension will seldom be more than 10-15 mm. Hg. I have had little or no personal experience with the Knapp adrenalin test, the jugular compression test, and some of the other less well known provocative tests.

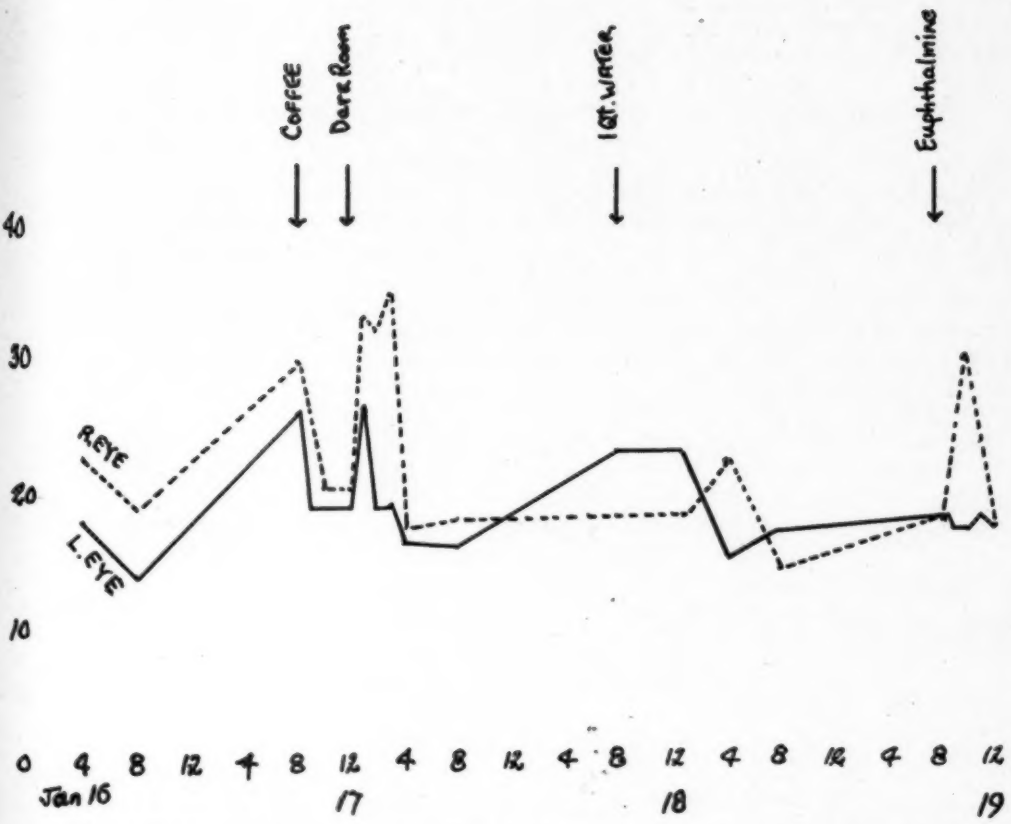


Fig. 1 (Gradle). Tension curve illustrating positive dark room and euphthalmin tests and negative coffee and drinking tests. Case of a thirty-five-year-old man with suspected glaucoma. Note the increased morning tension and the sharp rise in tension in right eye after one hour in dark room and after instillation of one drop of euphthalmin. Diagnosis of glaucoma was further corroborated two months later by continued high tension. No visual field, visual acuity, or ophthalmoscopic changes.

Eighteen years ago, Knapp of Basel brought to our attention the reduction of intra-ocular tension following digital massage of the eyeball. Two years later, I had tried this procedure upon a sufficiently large number of patients to be able to state that a reduction of less than 6 mm. Hg. in pressure by one minute deep massage was indicative of the approaching necessity of operative interference. I still believe that holds true. However the procedure is of little or no value in establishing the diagnosis in a doubtful or border-line case.

The curve of daily tension however is of greater diagnostic value than the provocative tests. In the normal eye, the daily variation in tension is seldom more than 6 mm. Hg. and particularly so when the patient is quiet in the hospital, apart from physical, gastronomic or mental excitation. The morning rise is well under the upper limits of the so-called normal tension, the apex of the rise seeming to be a more or less individualistic matter. But in a glaucomatous eye, the morning tension has risen to heights that are beyond the range of possible normal; that is to say, in the low forties. The descent from this level to the noon tension is apt to be quite abrupt so that the tension at midday could quite well be classified as high normal. During the afternoon, the decrease in pressure is more gradual as a result of which the late afternoon and the early evening

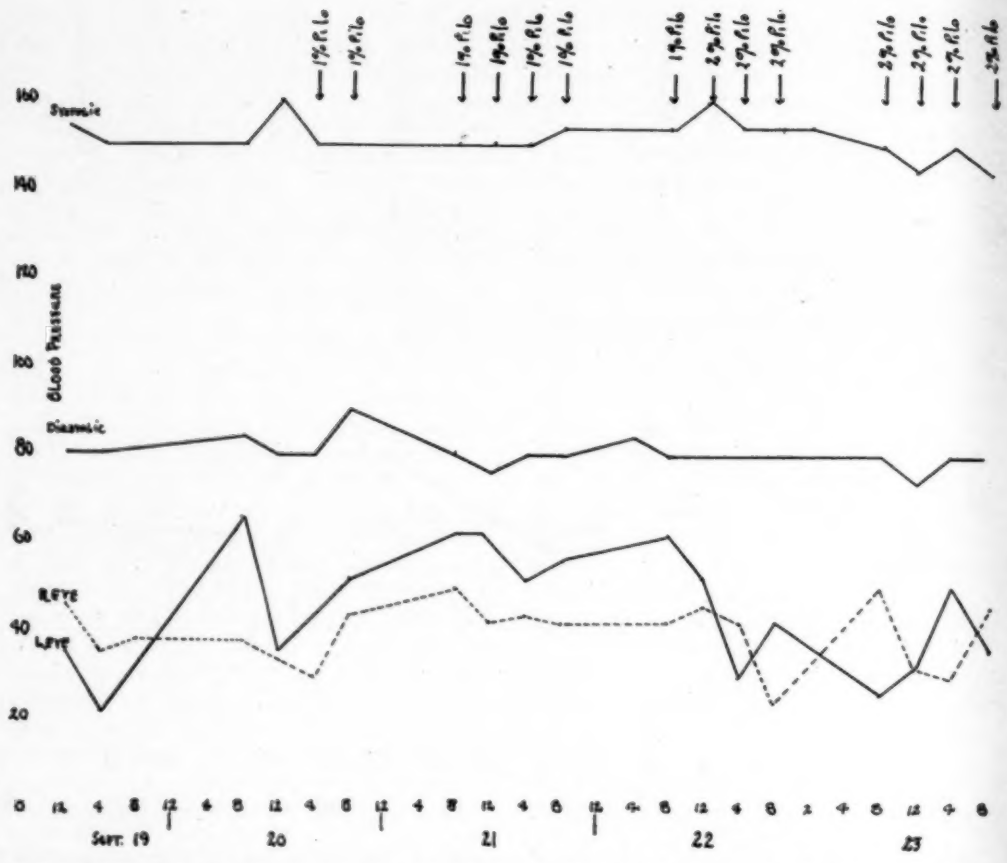


Fig. 2 (Gradle). Tension curve showing lack of relationship between blood pressure and intraocular pressure. The left eye has a chronic inflammatory glaucoma, probably following central vein thrombosis; the right eye has a noninflammatory glaucoma, fairly early, but difficult to control. Note that neither systolic nor diastolic pressure curves follow ocular tension curves in any respect.

tensions are likely to be about alike, both well within the range of normal. Thus it is quite apparent that the daily tension curve of an untreated glaucomatous eye does not parallel that of a non-glaucomatous eye, but on the contrary presents a radically different curve, which is characteristic enough to allow of a diagnosis.

Thus far we have spoken glibly of "normal tension", but what is normal tension? Accepting the Schiötz No. 2 Tonometer as being the most accurate of our clinical instruments (naturally I am partial to my own modification of the Schiötz), normal intraocular tension is supposed to range from 15 mm. to 29 mm. Hg. But it is certain that all clinicians of experience have seen "compensated glaucoma without perceptible rise in tension" wherein the tension never rose above 24-25 mm. Hg. at any measurement, but the disease progressed with all the characteristics of a simple glaucoma. On the other hand, I have observed one particular case for twenty years, during the last seventeen of which the tension of the patient's one remaining eye has been consistently from 31 to 38 mm. Hg. and despite which there has not been one iota of loss of vision or of contraction of visual fields or of development of central field scotomata. So what is normal? Evidently, the normal is that degree of pressure which the tissues of the eye are able to withstand without impairment of function. The resistance

is thus a function of the individual and not an absolute measure. The upper limits of that resistance can be determined in the individual case only by careful measurements when the pressure oversteps those limits.

If the various tests for incipient glaucoma have failed to give positive evidence of the disease and after the four hour curve has been followed for at least forty-eight hours without establishing a definite diagnosis, the pupil of each eye should be dilated with two per cent euphthalmin solution. The tension should then be measured every half hour for at least three hours at the end of which time a miotic should be used, merely as a precautionary measure. Euphthalmin is to be preferred to the other mydriatics for it produces a fairly prompt dilatation of the pupil and the effect can be controlled, should hypertension develop. But if the tension remains within the so-called normal limits, it may be stated definitely that the eye is not in a glaucomatous nor a preglaucomatous state. It stands to reason that the use of euphthalmin for diagnosis in a suspicious case must be limited to those cases where other provocative tests have all been negative and where a clinical diagnosis of glaucoma cannot be made. Again it must be emphasized that the total number of cases where the various provocative tests, including the use of euphthalmin, are necessary to establish a diagnosis of simple glaucoma, is small, for by the time the average case comes to the oculist, the diagnosis is manifest.

Hospitalization of a definitely diag-

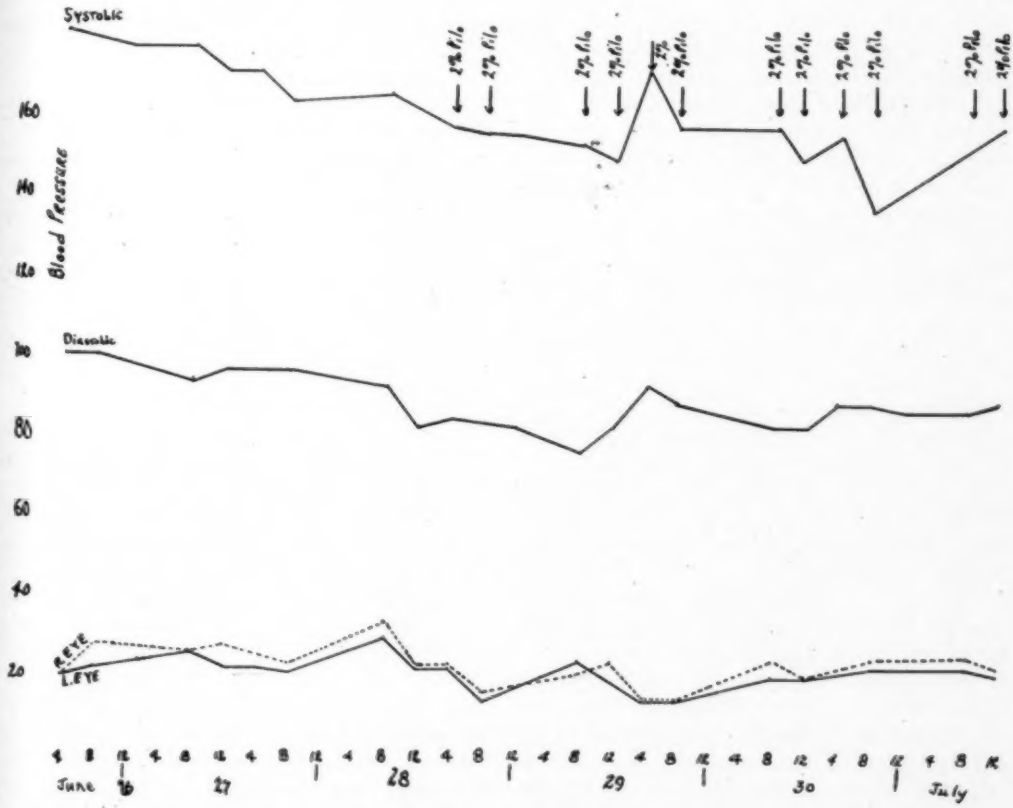


Fig. 3 (Gradle). Typical case of early simple glaucoma in a fifty-year-old woman. Note the early morning rise of ocular pressure on June 27 and 28, which was flattened out by pilocarpine on following days. Note the absolute lack of relationship between amount of blood pressure and amount of ocular pressure, particularly afternoon of June 28. The decrease in blood pressure was undoubtedly due to bed rest.

nosed case of compensated glaucoma is, to my mind, even more essential than in the merely suspicious cases. For only there can the reaction of the eye to the treatment that has to be instituted, be observed accurately. At this point, allow me to disagree most emphatically with Parsons who made the following statement in the last edition of his textbook (speaking of chronic or simple glaucoma). "An operation should be performed as soon as the condition is definitely diagnosed". The majority of ophthalmologists with glaucoma experience have numerous cases that have been carried for many years with miotics alone. Offhand, I can recall one case carried twenty-eight years, one twenty years, and one seventeen years, by the careful and controlled use of miotics, without sufficient impairment of visual function to handicap them or necessitate operation. It is true that a large percentage of compensated glaucomas eventually require surgical interference, but surgery must not be resorted to until it has been proven that the eye, aided by modern therapeutics, cannot hold its own against the ravages of the disease.

The therapy of simple glaucoma consists in the use of pilocarpine, and I make that statement advisedly. For the eye with a simple glaucoma that requires the constant use of eserine for the control of tension is an eye that requires surgical aid. The effect of pilocarpine may be enhanced from time to time as required, by the use of adrenaline, laevoglaukosan, gynergen, pacyl, or whatever means the surgeon has found most beneficial in the temporary reduction of tension. But that phase of the treatment of glaucoma is a separate subject that I do not intend to touch upon at this time.

After the diagnosis of compensated glaucoma has been established, the use of pilocarpine should be started. The strength employed and the frequency of instillation must depend upon the severity of the condition, a matter of judgment on the part of the surgeon. If the disease is in its incipiency, 0.5 percent pilocarpine used two or three times daily, may suffice to hold the daily tension curve within the range of normal. But if the morning rise approximates the curve of an eye with uncontrolled hypertension, the strength of the drug and the frequency of instillation must be increased until it can be determined whether or not the tension curve can be flattened by pilocarpine alone. If it can be, we are then in a position to instruct the patient how strong a solution of pilocarpine must be used and exactly at what hours of the day, or night. But if the morning rise continues to be unpleasantly high, eserine in one-fourth per cent strength should be added to the daily therapeutic pabulum and continued until the surgeon knows exactly how the eye responds.

Cases do occur in which a reverse effect of pilocarpine takes place. In a normal eye, the instillation of pilocarpine is followed by a slight and merely temporary rise in tension. As soon as the pupillary contraction is well under way, the tension sinks to below the premiotic level, but occasionally, the tension rises instead of sinking after the use of pilocarpine. I have had this happen in four individuals at widely separated intervals of time. In each instance, the pilocarpine was used in other individuals, both before and afterwards, without producing untoward effect, thus eliminating the possibility of jaborin impurity. Lilienfeld has shown that impure pilocarpine may contain jaborin, an isomeric form of pilocarpine which is really a mydriatic. In all of my cases, the increased tension was accompanied by a moderate dilatation of the pupil and a marked increase in tension, once amounting to practically an attack of inflammatory glaucoma. In every case, eserine was used, promptly contracting the pupil and reducing the tension. Within twenty-four hours, the patients were put back on pilocarpine and the usual miosis occurred without untoward incident. In a verbal communication, Dr. Benedict of Rochester told me that he had had a similar occurrence on two occasions. I have no explanation to offer for the phenomenon.

Although the surgical management

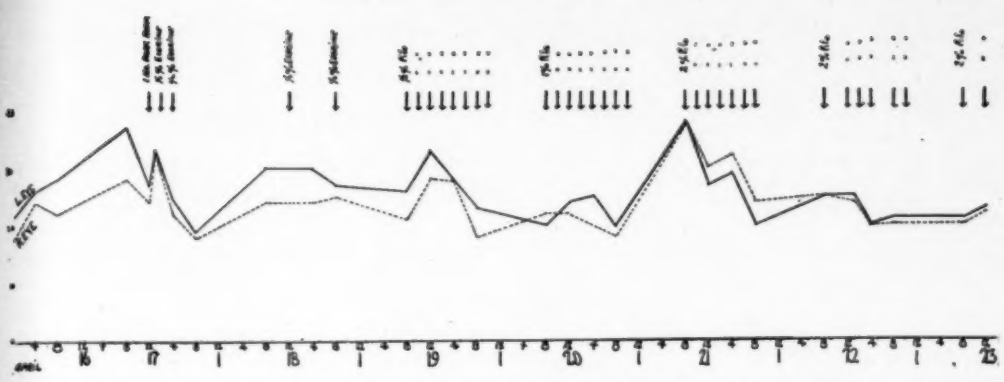


Fig. 4 (Gradle). Tension curve of an untreated case of glaucoma simplex in a forty-nine-year-old man. Note the morning rise on April 17, and subsequent decrease without miotics. Note the positive dark room test on April 17. Note the inverse pilocarpine reaction between 8 and 12 o'clock on the morning of April 19. Note the morning rise on April 21, showing that 1% pilocarpine was not strong enough to keep the tension normal for 12 hours. Such a rise never occurred under 2% pilocarpine. The tension could not be controlled with miotics and eventually it became necessary to perform bilateral cyclodialysis.

of glaucoma is not to be included in this paper, I believe that one new surgical fact can be reported here to advantage. It is a rather common experience that after a technically perfect iridectomy, hypertension may recur within one to three weeks, more frequently where the operation has been performed to relieve chronic inflammatory glaucoma rather than chronic simple glaucoma. In such cases, a cyclodialysis at the site of the iridectomy is almost uniformly successful in normalizing the tension. As the conjunctiva is apt to be tied down by adhesions, a more peripheral conjunctival section than the usual is needed. The scleral incision should be between five and six millimeters from the limbus and only long enough to admit the spatula. There is bound to be some little difficulty in working the spatula between the ciliary body and the sclera until the tip appears in the anterior chamber. Care must be taken in making the lateral sweeps with the spatula to free both pillars of the coloboma for it is very probable that therein lies the success of the procedure. It might be added that the operation is apt to be more painful than a primary cyclodialysis.

I suppose that there is more research today on simple glaucoma than on nearly all other questions in ophthalmology combined. And from all this work, some rather extravagant claims of therapeutic procedures are being advocated every other day, the majority of which are discarded within a fairly short time. Coincidental with these claims are coming the very definite refinements in diagnosis, some of which I have here discussed, and all of which lead to a better understanding of the subject.

58 East Washington street.

A GLAUCOMA STUDY

MICHAEL GOLDENBURG, M.D., F.A.C.S.
CHICAGO

A study was made of fourteen patients, suffering from different types of glaucoma, with especial reference to the permeability of the capillaries. All showed increased response to stimulation. The conclusion is drawn that the inherent cause of glaucoma lies in increased permeability of the capillaries of the ciliary body. The tabulated data of this study have previously been printed in the *Archives of Pathology* and are reprinted here, by permission, in order that the data may be more readily available in ophthalmic literature. Read before the Chicago Ophthalmological Society, April 20, 1931.

In 1927[1] I presented before this society, a theory on the mechanism involved in the production and maintenance of normal intraocular pressure. This was followed by a paper on the closure of the drainage angle[2]. The development of this viewpoint is the result of an inability to reconcile clinical observation and surgical experience with the traditional explanation offered for the various phenomena involved in the glaucoma problem.

As a matter of fact, the theory on the closure of the drainage angle was developed first, and after playing with that idea for a very long time, I came to the conclusion, that if the principle involved was sound, it must also explain the phenomena associated with the normal intraocular pressure. This was found to be true, in so far as one could accept theory for fact, that is, all the questions involved could be better answered on this basis than on the long existing teaching, which is likewise based on theory.

The viewpoint brought out in the previous papers, although apparently radical at the time, would seem to be justified in view of the fact that very little if any practical progress has been made in solving the problem since the time of von Graefe and de Wecker. It is therefore reasonable to presume that if the problem were studied from a different angle and investigation carried along such channels, possibly, as outlined in this work, some information may be developed that would be of real value.

The theories presented in these two papers are based on the newer concept of the anatomy and physiology of capillaries as propounded by Krogh and others and our present knowledge of the source and chemistry of the body fluids. The work in this field is now very extensive and authentic and lends itself happily to the explanation of many disputed phenomena.

The hypothesis advanced here is that in glaucoma, alterations in capillary permeability exist (which may be congenital or secondary to a stimulus), that result in abnormal or pathologic chemistry of the intraocular fluids. The very delicate sensitive anatomic structure peculiar to the eye, particularly the transparent media, lends itself favorably to the manifestation of symptoms which could pass unnoticed in any other part of the body.

Duke-Elder[3], Magitot[4], and others have demonstrated beyond question that the intraocular fluids originate from the blood stream within the eye ball. The former in particular has shown that every element found in the aqueous is also found in the blood stream and inversely that every soluble element found in the blood stream can be demonstrated in the aqueous. In the pathologic states the same facts have been found to be true, but in abnormal ratios. One may add that at times, even insoluble elements, for example, blood corpuscles may be found in the pathologic aqueous.

That capillaries are permeable has been known for many years. That they have the ability to contract and dilate has only recently been demonstrated by Krogh[5] and his followers. It was argued when my first paper on this subject was presented, that the Rouget cells, which in reality are a modified muscle layer and are found external to the endothelial tube of the capillary, could not be

demonstrated in the capillaries of the human eye, although at the time, their existence had been assumed, owing to their proven presence in other parts of the body. They have since been conclusively demonstrated by Schaly of Arnheim, Holland[6].

The nervous mechanism involved is the autonomic, that is, the sympathetic and parasympathetic systems. Of the sympathetic system we possess much information, at least anatomic, of the parasympathetic we cannot say so much. For example it has never been proven anatomically that this system exists, although pharmocologists contend that it must exist or certain known drug reactions could not be obtained.

Canon[7] and others have demonstrated some startling phenomena, which would appear pertinent to this problem. He has shown that an animal that is desympathectomized can carry on the normal functions of the body for a very long time without any apparent ill effects. But if this animal is subjected to extraordinary stresses, such as exposure to marked temperature changes, the animal will die, whereas the normal animal suffers no harmful effects.

That the autonomic system is an important factor in the control of capillary permeability is probably true, that is, during the cycle of relatively normal events. But when an element enters the circulation of such character as to produce a violent reaction, this system becomes disabled and cannot function as a stabilizer. One might draw an analogy between this system and a modern radio receiving set,—of what value is the refined mechanism for bringing out delicate tone shading in the presence of pronounced static? The damage sustained as a result of this violent reaction must return to a state of sufficient recovery before the autonomic system can again function as a stabilizer.

It has been shown by Petersen and others that if a capillary is stimulated by any means capable of such action, dilatation of the capillary follows and with it increased permeability. If this stimulation is continued, dilatation increases and with it greater permeability until the point of fatigue is reached, when the capillary becomes widely dilated and permeability reaches a maximum state. If the stimulation ceases before the state of fatigue is reached the capillary will return to normal, will contract and the permeability diminish.

It is on these several demonstrated factors that this theory is based. When the capillaries of the intraocular structures become excessively dilated, particularly those of the ciliary processes, permeability increases. Due to this increased capillary permeability, elements which are normally found in minute quantities in the aqueous, pass freely through the capillary wall into the fluid chambers. The resultant pathologic fluids in these chambers present, not only an unusual physical, but an unusual chemical state, which is followed by stasis, congestion and elevation of intraocular pressure. It is also quite likely that owing to the dilatation of the capillaries of the ciliary processes, the so-called drainage angle is physically encroached upon and this stasis and congestion accentuated further.

Magitot is of the opinion that the aqueous humor is a stagnant fluid and that the canal of Schlemm does not function as a drainage avenue. I cannot wholly agree with Magitot, but I certainly must disagree with Thompson's theory of the mode of action of this canal, the so-called pump action theory. I am inclined to think that the canal of Schlemm acts as a sort of safety valve or accessory avenue of fluid escape. By that I mean, if the capillaries are unable fully to take care of the disturbed fluid balance existing, the canal acts as an emergency avenue of escape, the meshwork of the drainage angle acting as a dialyzing membrane, through which the fluids must pass into the canal of Schlemm.

If we will dismiss the idea of the canal of Schlemm opening and closing according to Thompson's idea we have only the reasoning here described left to account for the function of this canal. To dismiss the pump action hypothesis it is only necessary to study this region anatomically antero-posteriorly instead

Table 1

THE CLINICAL HISTORIES OF PATIENTS WITH GLAUCOMA

Case	Sex	Age	Occupation	Clinical History	Vision	Tension (Schiötz)	Type	Operation
137	F	61	Housewife	2/3/27; pain in right eye for week; headache above orbit; lachrimation and photophobia; lids normal; conjunctiva injected; iris normal; pupil contracted; cornea cloudy and stippled; anterior chamber shallow.	R. hand move at 18" L. 20/30	R. 65 L. 15	Acute congestive. (inflammatory)	2/8/27 iridotasis.
140	M	45	Laborer	One year ago left eye was painful, but recovered without treatment; 9 months ago foreign body in left eye—altho eye was not very sore, vision was bad; 3 wks. ago eye was so painful patient had to stop work; eye injected, pupil dilated, cornea steamy and insensitive; hay fever.	R. 20/25 L. no light perception or projection.	R. 12½ L. 45½	Acute attack superimposed on chronic congestive glaucoma.	8/16/27 iridotasis.
141	F	48	Housewife	Trouble in left eye 3 or 4 years ago; vision gradually failed; not always painful; pain severe now and located in top of head; R. anterior chamber shallow; pupil dilated and not reactive to light; marked cup; L. scleral staphyloma at 12o'clock; iris drawn into staphyloma pupil oval and not reactive to light; anterior chamber shallow; iris cloudy; fundus visible.	R. 20/100 L. hand move.	R. 36½ L. 50¾	Chronic congestive.	Trephine.
145	M	71	None	Gradually failing vision for about 4 or 5 months.	R. 20/100 L. Fingers at 3 feet.	R. 16 L. 40	L. non-congestive. (Simplex)	8/2/27 iridotasis.
148	F	67	None	Chronic glaucoma in both eyes for several years; right operated on 3 years ago, became worse and later enucleated; left pupil dilated; cornea steamy; (vitiligo); old cerebrohemorrhage.	R. Enucleated, L. hand move.	L. 32	Chronic congestive.	9/6/27 iridotasis.
150	M	67	Teamster	Right enucleated; left hurt 3 weeks ago with iron object; became blind in 3 days; later cleared up; later, vision became foggy; some pain; L. slight injection of conjunctiva; cornea and iris normal; pupil regular; anterior chamber shallow; deep cupping of disc.	R. Enucleated. L. 20/50	L. 54	Non congestive.	3/1/27 iridotasis.
153	M	75	Farmer	Vision slowly failing for past 4 or 5 years; no pain; right pupil dilated, cornea steamy; anterior chamber shallow; left pupil dilated; anterior chamber shallow; cataract.	R. Perception and projection poor with bright light. L. Very poor with bright light.	R. 52 L. 48	Congestive. L. non-congestive.	L. 9/20/27 iridotasis of both eyes.
154	M	55	Laborer	Severe pain over right eye 2 weeks ago; similar attack 2 years ago; vision good until 2 weeks ago; then it began to fail with pain in head; conjunctiva congested; some ciliary injection; cornea hazy; pupil does not react to light; anterior chamber fairly normal.	R. 20/200	R. 61	Acute congestive.	10/28/27 Trephine.
160	M	58	Laborer	"Terrible" pain over right eye; patient unable to see; asthma.	R. Fingers at 4 ft. L. 20/40	.....	Acute congestive.	9/12/27 deep iridectomy. 10/18/27 iridectomy enlarged.
161	F	43	Housewife	Vision failing for past 3 or 4 years; occasional pain on right side of head. Pupil widely dilated; anterior chamber shallow and lens hazy; fundus indistinct; vessels of sclera dark; tortuous; marked cup; optic atrophy. Left normal, scleral vessels dilated.	.....	R. 50 L. 20	Chronic congestive.	10/18/27 iridotasis. 12/6/27 L. iridotasis.
164	M	48	Insurance agent	Pain last 4 months in R. eye and side of head. Pupil dilated; cornea hazy; anterior chamber deep.	.....	R. 42¾ L. normal.	Acute congestive probably secondary.	9/29/27 anterior sclerotomy 11/1/27 iridotasis.
167	F	77	Housewife	Vision failing for years; under care of Christian Science Practitioner; right cornea abraded, insensitive, opacity of lens; left the same.	R. light perception L. same	R. 16 L. 42¾	R. cataract. L. cataract with tension congestive glaucoma.	11/15/27 iridotasis.

Table 1 (Cont.)

Case	Sex	Age	Occupation	Clinical History	Vision	Tension (Schiötz)	Type	Operation
165	M	52	Laborer	Vision failing for year; occasional pain along side of head. Both pupils dilated; anterior chambers shallow; corneas stippled.	R. 2/200 L. 1/200	R. 70 L. 70	R. congestive. L. congestive.	10/18/27 iridotasis, both eyes.
170	F	56	None	Vision in left eye began failing in June, 1926; in September vision in left eye completely lost; vision in right eye began failing about the same time and was lost in April, 1927. Anterior chamber fairly good; pupils dilated eccentrically; new vessels formed on iris; some post synechia; cornea hazy; arcus senilis marked; opacity of both lenses.	No light perception in either eye.	R. 80¼ L. 85½	Congestive.	12/13/27 iridotasis right eye. 1/17/28 iridotasis left eye.

of from the usual sagittal section. We shall then find that the so-called scleral spur cannot possibly move back and forth as this theory suggests. Thus the meshwork of the drainage angle, composed of trabecular tissues and lined by endothelium remains as the barrier through which the fluids must pass to reach the canal. This can only be accomplished by a process of filtration, osmosis, or dialysis and in view of the lack of cellular findings in the canal of Schlemm one may conclude that the process of fluid escape into the canal is similar to that of its entrance into the chambers, namely by dialysis.

In regard to the stagnant state of the aqueous, we must visualize what Magitot meant and what we have understood by aqueous flow. According to the usual concept as propounded by Priestly Smith and others, so many cu. mm. are secreted, or flow, into the chambers and escape through the canal of Schlemm in a given length of time. The deductions made from the experiments conducted for proving this, although estimable at the time, must be considered today as faulty and the experiments themselves very crude. At best, they only disclosed reaction to abnormal stimulation. Emptying the anterior chamber is a very violent stimulant and a like reaction must follow. The same may be said of intraocular or intravascular injections of foreign substances, such as dyes, and recording the time in which they are eliminated from the eye. Such foreign substances act as irritants and the reactions following cannot be compared to normal physiological processes.

That the aqueous has some kind of motion, one may concede; witness, that due to thermal states and that possibly caused by the whipping of the aqueous during iris contraction and dilatation, but I do not think a regular flow into and out of the eye ball exists. What I do believe exists is a fluid interchange between the capillaries and the aqueous, and in addition a like interchange between the aqueous and the canal of Schlemm by a process of dialysis. Magitot is of a similar opinion, except he does not concede such function to the canal of Schlemm.

That the endocrines must be considered seriously when we speak of capillary permeability is evident to most workers. It is, of course, unfortunate that our knowledge of these various elements and their relation to each other is limited. That they play a part in the glaucoma problem has frequently been suggested, but is as yet to be demonstrated. For example, it is known that capillary permeability is increased during the premenstrual and menstrual period[8]. Likewise, it is influenced seasonally, that is, an increase occurs in the spring.

It is significant to recall that the majority of glaucoma cases present themselves at about the time of life which is associated with marked endocrine disturbances, particularly in the female. I am sure that the prodromal symptoms of glaucoma have many times been flippantly dismissed by the gynecologist or general practitioner, as symptoms of the menopause. I have elicited such a history on several occasions.

Just what particular element precipitates this cycle of phenomena is still de-

Table 2

THE RESULTS OF CORRELATIVE EXAMINATION OF FOURTEEN PATIENTS WITH GLAUCOMA

Individual Number	Blister Time, Hours	Permeability of Capillaries, %	Inflammatory Index	Calcium, Mg.	Potassium, Mg.	K/C Ratio	Sugar, Mg.	Globulin, per cent	Wassermann Reaction	Kromayer Light Erythema Time, Hours	Basal Metabolic Rate	Skin Resistance (Electric)	Changes in Urine	Blood Pressure, Systolic	Blood Pressure Diastolic	Pulse Rate	Epinephrine Reaction, Pulse Pressure, %	Epinephrine Reaction, Pulse Pressure × Pulse Rate, %	CO_2 Combining Power
137	15	61	4	8.1	18.2	2.24	73	37	..	2	3.6	1.9	Trace of albumin	119	67	78	+ 7	+ 9	51.9
140	0	68	0	11.4	29.7	2.61	82	37	..	2½	..	0.45	Trace of albumin	100	70	68	+10	+10	55.6
141	..	..	..	9	28.2	3.13	86	..	..	2	..	0.2	+	118	78	84	+ 9	0	64.6
145	15	66	4.4	10.1	15.7	1.55	70	55	+++	2¾	+19	0.35	Trace of albumin	136	80	84	+43	+43	55.4
148	15	56	3.7	9.7	24.3	2.50	79	24	..	1½	+17	0.26	..	116	74	90	+ 8	+18	69.5
150	15	57	3.7	12	21.3	1.78	82	15	..	1½	+39	0.22	Hyaline casts	140	86	84	+ 6	+ 6	62.7
153	15	78	5	9	18.7	2.08	83	0	..	1½	+ 2	0.15	Granular casts	124	58	64	+10	+10	62.2
154	15	60	4	..	23.08	..	71	3	..	1½	+18	0.09	?	194	118	68	..	..	56.6
160	15	66	4.4	10.86	21.58	1.98	65	44	..	1⅛	+16	0.4	?	128	64	68	+ 8	+28	62.7
161	5	72	14	9.17	18.03	1.96	86	20	..	1¼	16	1.4	Trace of albumin	107	64	88	+25	+51	49.4
164	9	71	8	8.7	16.54	1.89	65	20	+++	2¾	− 4.7	0.23	Granular casts	110	76	80	+14	+35	55.9
165	11	60	5.5	10	17.94	1.79	80	16	..	1⅛	+ 0.5	0.18	Hyaline casts	170	100	110	..	..	53.9
167	15	56	3.7	12	22.72	1.9	81	27	..	2	0.7	0.85	..	137	92	84	62	80	47.9
170	15	72	5	10.58	23.71	2.2	320	36	..	..	+33	0.07	..	188	96	104	..	..	52.5
Averages*	13.3	64	4.8	10	21.4	2.14	77	25	2+	1 1/4	+12.4	0.48	..	134	80	82	+18	+29	57.2
Averages†	7	63.3	9.4	10.1	20.6	2.04	71.3	30		2	+ 6.3	0.35	..	116	69	71	+16.5	+24	58

* For 14 patients with glaucoma.
† For 37 normal persons.

Table 2 (Cont.)

COMPARISON OF THE AVERAGES OF THESE RESULTS WITH THE AVERAGES FOR THIRTY-SEVEN NORMAL PERSONS

Endothelial Reaction (Rumpel-Leede)	Roentgen Examination of Chest: Heart	Roentgen Examination of Chest: Tuberculosis	Capillaries of the Skin (Microscopic Examination)	Reaction to Ice, Seconds	Age	Weight/Length	Cholesterol, Mg.	Intracutaneous Reactions: Epinephrine, Wheal, Mm.	Epinephrine, Flare, Mm.	Morphine, Wheal, Mm.	Morphine, Flare, Mm.	Thyroxin, Wheal, Mm.	Thyroxin, Flare, Mm.	Caffein, Wheal, Mm.	Caffein, Flare, Mm.	X-Ray Diagnosis
..	Asthenic	..	Dilatation	30	61	166	222	15.4	11	13.5	10	13	0	18	0	
..	Asthenic	+	..	40	45	..	158	18	8	11	10.7	27	0	30	0	Active tuberculosis of right upper lobe
+	Sthenic	..	Granulation	15	48	..	267	17.4	7.7	9	7	10	13	22	0	
..	Sthenic	..	Granulation	20	71	166	208	18	13.6	16	0	16	0	23	0	Syphilitic aortitis
..	Asthenic	Fibrosis	Granulation	10	67	182	317	18	12	9.7	0	17	0	18	0	Arteriosclerosis (aorta)
..	Sthenic	Fibrosis	..	7	67	238	247	17	14	18.5	9.5	16	0	22	0	Pulmonary fibrosis
..	Sthenic	Healed parenchymal	Diminished dilatation	30	75	223	175	22	6.5	9.5	0	18	0	22	0	Slight arteriosclerosis; fluid in chest
..	Asthenic	..	Granulation	20	55	181	180	23	10	13.5	0	26	0	28	0	Arteriosclerosis (aorta)
..	Sthenic	Healed parenchymal; fibrosis	Normal	10	58	181	190	16	8	12	0	20	0	24	0	Arteriosclerosis (aorta); bronchiectasis; emphysema
..	Sthenic	..	Dilatation	12	43	171	208	14	13	5.5	10.7	13	0	20	0	
..	Sthenic	Healed parenchymal	..	15	48	223	185	18	14.7	17	12.5		(Not done)			Syphilitic aortitis
..	Sthenic	..	..	5	52	250	185	17	13	10	13.7	25	0	31	0	Hypertrophy of heart; dilatation of arota
..	Asthenic	..	Dilatation	5	77	158	256	17	14.25	12	9	15	0	18	0	Scoliosis; bronchiectasis
..	Sthenic	..	..	..	56	247	170				(Not done)					Arteriosclerosis (aorta); bronchiectasis
..	..	..	..	15.6	58.7	199	212	17.7	11.2	12.1	6.4	18	1	23	0	
..	..	..	..	15.5	41.8	221	228	20	9	11.3	16.2	11.4	5.8	17.4	2.38	

batable, but one may assume that some irritant or stimulant is present in the blood stream.

Discussing the theories presented in the papers referred to and the part played by the capillaries, with Doctors Petersen and Levinson resulted in an invitation to refer to them and their associates, a series of glaucoma cases for study, as a part of the various pathologic groups under observation at that time. Their work consisted of a study of one hundred pathologic cases, among which this group of fourteen cases of glaucoma was included. These studies were then compared with one hundred so-called normals previously studied under similar conditions[9].

When we so analyzed our small series we found that every one presented definite evidence of alteration referable to the vascular system.

Of the two patients who had syphilis, No. 145 had distinct granulations in the capillaries of the skin, albumin in the urine and, as revealed by an x-ray picture of the chest, syphilitic aortitis; No. 164 had granular casts in the urine, and the x-ray picture revealed syphilitic aortitis.

Of the three patients who had high blood pressure, No. 154, whose blood pressure was 190 systolic and 118 diastolic, revealed an arteriosclerosis of the aorta on roentgen examination, and granulations of the capillaries of the skin. No. 170, whose blood pressure was 188 systolic and 96 diastolic, had a blood sugar content of 320 mg., a basal metabolic rate of + 33, and a pulse rate of 104. The roentgen examination showed a definite sclerosis of the aorta. No. 165, whose blood pressure was 170 systolic and 100 diastolic, had hyaline casts in the urine and a pulse rate of 110; roentgen examination showed an enlarged heart and a dilated aorta. The rest of the patients had blood pressure within the normal range. Of these, No. 150 had hyaline casts in the urine and gave roentgenologic evidence of pulmonary fibrosis. No. 153 had granular casts in the urine and gave roentgenologic evidence of arteriosclerosis, as well as fluid in the chest. No. 137 had albumin in the urine, and the capillaries of the skin showed distinct dilatations. No. 140 had albumin in the urine and suffered from hay fever; the roentgenogram of the chest indicated some activity at the right upper apex. No. 141 had albumin in the urine, and the capillaries of the skin showed marked granulations. No. 161 had albumin in the urine, and the capillaries of the skin showed marked dilatations. No. 148 gave a history of cerebral hemorrhage; the roentgenogram indicated a marked sclerosis of the larger thoracic vessels, and the capillaries of the skin showed marked granulation. No. 160 had asthma; in addition, the roentgenogram indicated a dilatation of the aorta, with arteriosclerosis, and fibrosis and atelectasis of the lungs. No. 167 had low CO_2 combining power, in addition to the highest sensitivity to epinephrine observed in the group. The capillaries of the skin revealed no marked changes. The roentgen examination indicated an old pulmonary fibrosis and bronchiectasis.

The details of the study are collected in Table 2. Immediately below the averages of these details we have tabulated the averages obtained in examinations of thirty-seven normal persons, for comparison. These thirty-seven were normal persons and persons with roentgenologic evidence of healed parenchymal tuberculosis (part 2).

In all the cases of this small series, we have established that there are definite vascular alterations, in the form of urinary changes, changes in the capillaries of the skin, increased blood pressure, sclerosis of the larger thoracic vessels, or a history of disease known to be associated with autonomic vascular disturbances (asthma, hay fever).

The intracutaneous reactions may also be interpreted in this direction. Thus, the flare of the reaction to morphine is diminished, and the flare of the reaction to caffein, and that of the reaction to thyroxin are absent. The wheal in the reaction to epinephrin, on the other hand, is diminished. The wheal in the reaction to thyroxin is increased.

Résumé

The outstanding defect in this study is very apparent, that is, the small num-

ber of cases and the exhaustiveness of this study. If the investigation had been more limited, some of the defects would probably not have been disclosed and deductions made accordingly. Nearly all of them show changes that are referable to the cardiovascular renal system, but the youngest is 43 years old and the oldest 77 years; and such changes may be reasonably expected at this time of life.

In the papers referred to (no. 1 and no. 2) the conclusions reached were, that the inherent cause of glaucoma lies in alterations in the capillaries, particularly of the ciliary body, these alterations consisting of an increase in permeability, probably associated with an increased metabolic activity of the cells of the ciliary body, with a resulting swelling of this body, thus encroaching on the avenues leading to the canal of Schlemm.

Petersen and Levinson state[9], "It is our opinion that these alterations may take place rapidly and particularly in persons who are constitutionally so oriented. That the vessels of the eye may show such sudden swelling of the endothelium, has been observed by Lenaz[10] in a case in which the endothelial occlusion in an arterial branch was so great that an embolus was suspected."

Endothelial instability is found associated with certain endocrine or autonomic disturbances, which only in recent years have been studied more intensively. In the group showing this association, are included persons with urticaria, angioneurotic edema, hay fever, asthma, migraine and mucous colitis, conditions in all of which distinct alterations may occur in the ionic equilibrium (particularly the K/Ca ratio, CO_2 combining power, cholesterol content, and so forth), alterations not yet fully explainable. To this group must be added the persons with hyperpiesis, as well as the ones with hypotension, which according to Kylin[11] are merely different clinical responses to the same basic alteration.

In view of these deductions, one must give due consideration to the congenital or inherited status of these systems. The part played by the endocrine system may be of much importance. It may be argued that this system influences the autonomic system or vice versa.

Again, if a conclusion may be drawn from Canon's work, it must be, that an animal can carry on the normal functions of the body under fairly normal conditions without a sympathetic nervous system. Furthermore we know that glaucoma is comparatively rare in disease of the thyroid gland, which is rather common. Likewise is it true in disease of the pituitary bodies, adrenals, and the glands of sexual function. That disturbance of these two systems may produce an insidious process which could favor the precipitation of a glaucomatous attack is not unreasonable. However, this writer is of the opinion that the precipitating factor in glaucoma is in the blood stream. The factor may be variable, but it must be capable of producing a reaction in the capillary system that is beyond the immediate control of the sympathetic nervous system.

When applying this theory, due consideration must be given to the eye that is predisposed to glaucoma at birth. This is in itself a lengthy subject and must be left for another time, but one may say in passing that the so-called predisposing factors usually referred to, enlarged lens, hyperopia, and shallow anterior chamber may be dismissed. As a predisposing factor of import, one may give serious thought to a congenital tissue sensitivity in the capillaries, capable of a marked reaction to a stimulus.

104 S. Michigan boulevard.

References

[1] Goldenburg. Ill. Med. Jour., 1927, v. 52, p. 474.
[2] ——. Amer. Jour. Ophth., 1928, April.
[3] Duke-Elder. The Nature of the Intraocular Fluids, 1927.
[4] Magitot. Amer. Jour. Ophth., 1925, v. 8, p. 761.
[5] Krogh. Anatomy and Physiology of Capillaries. 1922-1929.

[8] Schaly, G. A. Groningen, Den Haag, The Rouget Cells on the Capillaries of the Human Eye. 1926.
[9] Canon. Lecture before the Institute of Medicine, Chicago, 1930.
[10] Petersen, W. F. Arch. Int. Med., 1926, v. 38, p. 730.
[11] Petersen and Levinson. Arch. of Path., 1930, January, v. 9, part 2.
[12] Lenaz, L. Wien. klin. Wchschr., 1926, v. 39, p. 1015.
[13] Kylin, E. Acta med. Scandinav., 1927, supp. 19 part 1.

EVALUATION OF PSEUDO-ISOCHROMATIC TESTS FOR THE DETECTION OF COLOR-BLINDNESS

J. ELLIS JENNINGS, M.D.
SAINT LOUIS

Since lower grades of color-blindness may be overlooked without careful examination, it is recommended that three tests be used in all cases. The Stilling, Edridge-Green, Ishihara, and Nagel tests are described. The results obtained with each test in examining a series of twenty cases of known color-blindness are tabulated and the tests evaluated. Read before the St. Louis Ophthalmic Society, February 27, 1931.

Like the finger-prints of individuals, no two cases of color-blindness are exactly alike, nor do the patients make the same mistakes when tested. Gross forms of red and green blindness are easily detected, but less marked cases may be overlooked unless a thorough examination is made with a number of different tests. There has been a great deal of discussion in this country and abroad as to the relative value of this or that test until the whole subject is in a state of confusion. The error lies in expecting any one test to be infallible. Why not select three of the best ones and use all three in testing every case? The three I would recommend are: 1. The Holmgren worsteds or the Jennings self-recording test. 2. The Williams or Edridge-Green lantern. 3. One of the pseudo-isochromatic tests.

In the *American Journal of Physiological Optics*, v. 5, no. 3, 1924, appeared a very interesting article entitled, "The Ishihara Test for Color-blindness," by J. H. Clark, Johns Hopkins, Baltimore. Clark examined two color-blind students with the Stilling and Ishihara tests, and four color-blind students with the Edridge-Green test and came to the following conclusions: "1. The Stilling test. Only a few of the groups in the Stilling test are practically useful, the others being both confusing and misleading, and some of the plates are almost impossible for the normal eye to distinguish. 2. The Edridge-Green test. Cards 3, 5, 6, and 22, indicate red-green blindness very consistently, but the entire set is unsuitable for general use, partly because the key that goes with the set is so very misleading (the Edridge-Green classification of color-blindness), and partly because some cards are difficult for the normal eye to read. 3. The Ishihara test. The Ingenuity and simplicity of the Ishihara test and the clear cut results obtained with it in practice make it to my mind the most successful of the three tests."

Before recommending one of the pseudo-isochromatic tests as my third test I decided to check the above findings with a much larger number of color-blind individuals. I also made a study of the Nagel test which is largely used in Germany. This report is based on the examination of twenty cases of color-blindness discovered among 5,021 railroad employes. Fifteen or twenty persons also with normal color sense were tested and note made of the plates which they found difficult to read.

1. **The Nagel test.** The ninth edition of Nagel's plates consists of 16 plates *A* and 4 plates *B*. On each plate is a ring of small round colored spots; one ring may be composed of spots of various shades of one color, red, green, brown, or gray, or a ring may have a diversified combination of those colors.

The color-blind is not able to distinguish the one colored rings from the multi-colored ones because the color hues have been chosen in such a manner that they are so-called "deceptive colors" for the color-blind. Most of the colored spots in division *A* plates appear to the color-blind as an indistinct color which they may take for gray. Out of indistinct gray of the mass of spots only those spots stand out more or less distinctly colored which are yellowish-green, or yellowish-brown which they mistake for red.

Method of examination. 1. The 16 plates of the *A* division are spread out on a table in good daylight and the following questions are put; 1. Can you show me some plates in which red or reddish spots are to be seen? 2. On which plate are only red spots? On which only green ones? 4. On which only gray ones?

II. After removal of the plates of division *A*, the examiner exhibits the four plates of division *B* singly. While keeping these in his hand, he asks what colors are to be seen in each.

The Nagel Test calls for great patience and alertness on the part of the examiner. Color names are used and the candidate is required to discriminate between pale shades of red, green, brown, and gray. Twelve patients of my series of 20 made mistakes with the Nagel test, calling gray green, brown green, and red green. The others hesitated and probably would have made some error had I exercised more patience and care. For some reason, confusing a pale gray spot with a pale green spot does not make the same impression on the mind of the examiner as when a gray skein is selected to match the green skein of the Holmgren test.

2. **The Edridge-Green test** consists of 24 cards 4¾ inches square, numbered on the back from 1 to 24. On 22 of the cards is a letter in spots of one color on a ground of spots of another color. On six of the cards are two letters, one enclosed within another like a monogram. In explaining the nature of the test Edridge-Green says, "A person of normal color vision can distinguish six colors; red, orange, yellow, green, blue, and violet; after this in descending order of acuteness of color vision, there are those who can distinguish five, four, three, two colors and finally the totally color-blind, who distinguish no colors and see the whole spectrum as a shaded gray band. I call those who can distinguish six colors hexachromic, and those of less acute perception in descending order, penta-, tetra-, tri-, and dichromic." This classification is quite at variance with the ordinary classification, red-green blind, blue blind, and totally color-blind.

Table I gives the results of testing 20 color-blind individuals with Edridge-Green's cards. In constructing the test a special diagram was made in which the shape, relative position and number of the spots was the same on all 22 cards. In consequence, some of the letters are irregular in shape and are difficult to recognize; for example, the letter B is often called 8. Cards 11 and 12 are rarely recognized by the normal eye. Cards 1, 2, 4, 7, 13, 15, and 19 are poor cards as they can be read by many color-blind persons. The six cards having two letters, one enclosed within another, are confusing as very few normal eyes see more than one letter unless their attention is drawn to the fact. The good cards which can be relied upon to detect red-green blindness are 3, 5, 6, 8, 9, 10, 14, 16, 17, 18, 20, 21, and 22, thirteen in all.

My study of the Edridge-Green cards shows it to be a very satisfactory test for the detection of color-blindness. No one in my series of twenty cases was able to read cards 3, 5, 9, 10, 14, 18, 21, 22. All of the normal individuals tested found the Edridge-Green letters more difficult to read than the numbers of Stilling, probably because of the more or less irregular shape of the letters. A point in favor of the Edridge-Green test is that the cards are not bound in book form and can be mixed up before beginning the test.

3. **Stilling's test.** The first pseudo-isochromatic plates were introduced by Stilling many years ago. With the as-

Table 1

EDRIDGE-GREEN'S CARDS

No. of Card	Color of Letter		Color of Background	Failure to read Indicates	Normal Eye	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	Read	Failed
1	Blue	M	Crimson	Shortening red end	M	–	–	M	–	–	–	M	–	M	M	–	–	M	M	M	M	–	–	–	–	8	12
2	Olive green	N	Red	Red-green blind	N	–	N	N	–	–	–	N	–	–	N	–	–	–	N	N	–	–	–	N	–	7	13
3	Orange	C	Yellow-green	Red-green blind	C	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
4	Yellow-brown	S	Orange-brown	Red-green blind	S	S	S	S	S	S	–	S	–	S	S	S	–	S	S	S	S	–	S	S	S	16	4
5	Crimson	L	Orange-brown	Shortening violet	L	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
6	Orange-brown	G	Olive green	Red-green blind	G	–	–	–	–	G	–	–	–	–	–	–	–	–	–	–	G	–	G	–	–	3	17
7	Red	N	Olive green	Red-green blind	N	–	N	N	–	–	–	N	–	–	N	–	–	–	N	N	–	–	N	–	–	7	13
8	Yellow-green	C	Orange	Red-green blind	C	–	–	C	–	–	–	–	–	–	C	–	–	–	–	–	–	–	–	–	–	2	18
8	Orange-brown	S		Pentachromics	?	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
9	Olive-green	G	Yellow-brown	Red-green blind	G	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
10	Yellow-green	N	Yellow-brown	Red-green blind	N	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
11	Orange-brown	P	Yellow-brown	Red-green blind	?	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
11	With green tail	R			?	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
12	Green	L	Gray	Red-green blind	?	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
12	Purple	E			?	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
13	Crimson	S	Green	Red-green blind	S	S	–	–	S	–	–	–	–	–	S	–	S	–	–	–	S	S	–	–	–	6	14
13	Yellow-green	G		Trichromics	?	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
14	Crimson	S	Blue-green	Red-green blind	S	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
14	Green	G		Trichromics	?	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
15	Blue	O	Green	Tetrachromics shorten violet	O	O	O	O	–	O	–	O	–	O	O	O	O	–	O	O	O	O	O	O	O	16	4
16	Red	R	Myrtle-green	Red-green blind	R	R	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	19
17	Red-orange	S	Green	Red-green blind shorten red end	S	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
18	Green	S	Orange-crimson and red	Red-green blind shorten red end	S?	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
19	Blue	B	Violet	Red-green shortening red or violet	B	–	B	–	–	B	–	–	–	–	–	–	–	–	B	–	–	B	B	–	–	5	15
20	Purple	B	Blue	Similar to 19	B	–	–	–	–	B	–	–	–	–	–	–	–	–	–	–	–	–	B	B	–	3	17
21	Green and olive	S	Orange-brown	Red-green	S	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
21	Red	O		Tetrachromics	O	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20
22	Orange-brown	M	Myrtle-green	Red-green blind	M	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0	20

Table 2

STILLING'S PLATES

Group	Color of Number	Color of Background	Failure to read Indicates	Normal eye	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	Read	Failed
1	Pinkish red	Bright green	Red-green blindness	+	+	−	+	+	+	+	−	−	−	+	+	+	+	+	−	+	+	−	+	+	14	6
II	Orange	Olive		+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	0	20
III	Red	Brown		+	+	+	+	+	+	+	+	+	+	+	+	+	−	+	+	+	+	+	−	+	18	2
IV	Red	Gray		+	−	+	−	−	−	−	−	+	+	+	+	−	−	+	−	−	−	−	−	+	7	13
V	Dull rose	Greenish gray		+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	0	20
VI	Red-brown	Greenish brown		+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	0	20
VII	Pink-brown	Mixed browns		+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	0	20
VIII	Red-brown	Brown and tan		+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	0	20
IX	Red	Orange-brown		+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	20	0
X	Olive	Blue	Yellow-blue blindness	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	20	0
XI	Green	Blue		+	+	+	+	+	+	+	−	−	+	+	+	+	+	+	+	+	+	+	+	+	18	2
XII	Green-orange	Rose gray	No marked color contrast	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	0	20
XIII	Red	Gray	Malingering	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	20	0
XIV	Red-brown	Tan and brown		+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	20	0

sistance of a red-green blind painter and a blue-yellow blind teacher he built up two classes of interchangeable colors. It was found that the red-green blind could not distinguish numbers in red or green shades on a background of browns, tans, and grays and vice versa. The seventeenth edition of Stilling's plates was published in 1926. It consists of 32 plates, with two sets of figures on each plate and is arranged in fourteen groups. There are from one to four plates in each group. Each plate consists of a number or numbers in spots of one color on a background of another color. Failure to read the numbers of groups I to IX indicates red-green blindness. Failure to read groups X and XI indicates yellow-blue blindness. Groups XIII and XIV can be read by color-blind individuals and are used to explain the test and also to detect malingering.

Table II gives the results of testing 20 normal and 20 color-blind individuals with Stilling's plates. Those with normal color-sense read all the plates except plate XII which the directions say is only seldom recognized by normal individuals. As none of the color-blind read this plate, it serves no useful purpose. Some of those with normal color sense hesitated over group VI, especially plates 1 and 3 in which there are three small numbers irregularly placed. The greatest interest centers in Groups I to IX in which a failure to read indicates red-green blindness. The best plates are groups II, V, VI, VII, and VIII, which none of the 20 color-blind could read. The next best is group IV which was read by 7 out of 20. The poorest are group I, read by 14 out of 20, group III, read by 18 out of 20, plate IX, read by all 20 and plate XII which neither the normal nor color-blind could read. My study of Stilling's test shows that if Groups I, III, IX, and XII are disregarded, there are 17 excellent plates to test red-green blindness, 4 plates to test yellow-blue blindness, and 4 plates to explain the test or detect malingering. I cannot agree with the statement of Clark that such a large number of plates is unnecessary and tends to confuse both the examiner and the subject. In testing the color sense it is necessary to have in reserve a large number of plates in order to detect varying degrees of color-blindness. Clark's conclusion that only a few of the groups in the Stilling test are practically useful, the others being both confusing and misleading, is not substantiated by my series of cases. It is unfair to condemn a test on the findings of two cases. For example, Clark recommends Group I because two color-blind persons failed to read it. I consider it a poor group because 14 out of 20 color-blind persons could read it.

The Ishihara Test. This is a Japanese test and consists of 16 plates, 13 of which have numbers in spots of one or more colors on a background of spots of another color. Four of the plates, 6, 7, 8, and 9, are similar to the Stilling plates and cannot be read by the red-green blind. In the other plates the color-blind see a number but a different number from that seen by the normal eye. For example, when shown a plate with the number six, the color-blind call it five, or the number 74 becomes to the color-blind a 21. On several cards a number is clearly seen by the color-blind but no number is visible to the normal eye. This new feature is based upon the fact that to the red-green blind, yellow, blue and violet are much brighter colors than red and green. Suppose the number 6 is built up in green and blue spots on an orange-brown background. Insert a few purplish spots which together with the blue form the number 5. To the normal eye the purple and blue spots are inconspicuous and the number 6 is clearly visible, but to the color-blind the green spots of the number fade into the background and he sees the number 5 made up of blue and purple spots.

Table III gives the results obtained with the Ishihara test. It will be seen that with the exception of Plate 2, read by five out of twenty, none of the other plates were read correctly by my series of twenty color-blind persons. Of great interest are two plates designed to discover whether a color-blind person is

Table 3

ISHIHARA TEST.

Page	Color of Number	Color of Background	Normal	Red Blind	Green Blind	Number read by patient																				Read	Failed
						1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20		
1	Red 12	Blue	12	12	12	12	12	12	12	12	12	12	12	12	12	12	12	12	12	12	12	12	12	12	20	0
2	Red and rose 8 Rose 3	Green	8	3	3	8	3	8	3	3	3	3	3	3	3	3	3	3	3	8	8	3	3	3	8	5	15
3	Red and rose 6 Rose 5	Green	6	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	0	20
4	Green and blue 5 Rose and blue 2	Orange-brown	5	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	0	20
5	Green and blue 74 Rose and blue 21	Orange-brown	74	21	21	21	21	21	21	21	21	21	21	21	21	21	21	21	21	21	21	21	21	21	21	0	20
6	Red 2	Green and blue	2	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	19
7	Red 6	Green and blue	6	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0	20
8	Greenish blue 5	Rose and orange	5	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0	20
9	Greenish blue 7	Rose and orange	7	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0	20
10	Violet and blue 5	Red and green	—	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	20	0
11	Violet and blue 2	Red and green	—	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	20	0
12	Red 2 Rose 6	Gray	26	6	2	6	2	6	2	2	2	2	2	2	2	2	2	2	2	2	2	6	2	2	2	3	Red blind
																										17	Green blind
13	Red 4 Rose 2	Gray	42	2	4	2	4	2	4	4	4	4	4	4	4	4	4	4	4	4	4	2	4	4	4	3	Red blind
																										17	Green blind

red or green blind. On one plate the normal eye sees the number 26, the red-blind see only the number 6 while the green-blind see only the number 2. When examined with these plates it would appear that only 3 of my cases were red-blind and 17 were green-blind. Were it not for one grave defect I would unhesitatingly endorse the Ishihara test as superior to the others. Officials are amazed and greatly impressed when an innocent number like 74 is metamorphosed by the color-blind into 21, but herein lies the weakness of the test. The number he sees furnishes him a key to the correct number. How long do you think it would take a man to learn that when he sees the number 21 he must call it 74; when he sees 3 to call it 8; when he sees 5 to call it 6; when he sees 2 to call it 5? The only good plates are 6, 7, 8, and 9 in which no number is seen by the color-blind, and these are similar to the Stilling plates. All the other plates are worthless because you never could be sure that the candidate had not memorized the correct numbers. For this reason the Ishihara test should not be used as the *sole method* of testing the color-sense in railroad or navy service. Color-blind men might buy the test and commit it to memory.

Conclusions

1. **Nagel's test.** This test requires the use of color names, which is a bad feature. In the discrimination of pale shades of red, green, brown, and gray, too much is left to the judgment of examiner and candidate. While it is a useful test for the expert, I do not think it would give reliable results in the hands of the ordinary examiner.

2. **The Edridge-Green** test is a very satisfactory test for the detection of red-green blindness if his classification of the color-blind is ignored. From the fact that some of the letters are more or less irregular in shape, they are more difficult to read than numbers. Cards which are difficult for the normal eye to read should be removed.

3. **The Ishihara test.** For the reason that this test can be committed to memory, it should not be used as the *sole method* of testing the color sense in railroad or navy service where a failure to pass means the loss of a job.

4. **The Stilling test.** In this test are 17 plates in six groups which are excellent for the detection of red-green blindness. Of the four tests my preference is for Stilling's, and I would therefore recommend it as a third test in association with the worsteds and the lantern.

5. The fact that certain cards are read does not mean that the candidate is not color-blind. It may be the fault of the card.

6. It is important to remember that in using any of these tests the plates are not to be shown in regular order.

All of the above tests may be had of C. H. Stoelting Co., 424 North Homan avenue, Chicago.

807 Carleton building.

NOTES, CASES, INSTRUMENTS

DIPLOPIA CORRECTED BY HIGH DEGREE PRISMS

A. F. Luhr, M.D.
BUFFALO

E. H., a girl aged 13 years, was first seen by me in November, 1927. Her complaint was of blurred vision. There was vertical diplopia and head tilting to left, which corrected her diplopia to some extent. She had been attending a private school and was always at the head of her class. For the past two years prior to her first visit to me she had kept one eye closed to avoid double vision. Her mother had had diplopia at the age of fifteen years and the mother's father had had the same condition at the age of thirty years.

Examination: Vision O. D. was 20/30; O. S. 20/30+3. Refraction under homatropine revealed an astigmatism corrected by +0.37 cyl. ax. 90°. This gave a vision of 20/20 in each eye. Tests of muscle balance showed a left hyperphoria of 16 degrees. She was given the following correction:

O.D. + 0.37 cyl. ax. 90° with 6 degree prism base up.

O.S. + 0.37 cyl. ax. 90° with 5 degree prism base down.

This correction gave her binocular single vision and comfort until March, 1929, at which time she complained of some headaches and a tendency to diplopia. The only change was a slight increase in the astigmatism and in the hyperphoria. She was given the following with relief:

O.D. no change.

O.S. + 0.50 cyl. ax. 90° with 6 degree prism base down.

In October, 1930, one and a half years later, she again had a marked diplopia with symptoms of strain. On examination I found the astigmatism had increased and the left hyperphoria was 21 degrees. The parents at this time objected to an operation and the patient was contented to wear the heavy prisms. The following correction was prescribed:

O.D. + 0.75 cyl. ax. 90° with 8 degree prism base up.

O.S. + 0.75 cyl. ax. 90° with 9 degree prism base down.

The patient returned within ten days relieved of ocular symptoms. Some time later however the symptoms did recur and the hyperphoria had increased to 25 degrees. The prisms were not changed but a three degree grab was given, a total of 20 degrees. She is again comfortable.

The interesting part of this case is the steady increase in the hyperphoria of a high degree with relief of her symptoms by prisms and the fact that the mother and mother's father also had this trouble.

40 North street.

ENUCLEATION SNARE

C. W. Banner, M.D., F.A.C.S.
GREENSBORO, N.C.

For several years, it has been my practice, in enucleating an eyeball, to use a tonsil snare for cutting the optic nerve and vessels at the posterior part of the eyeball. This was suggested by Dr. O. W. Turner. In cases where there had been ulceration or the tissues were otherwise weakened, I found that the tug on the eyeball in closing the snare was apt to produce rupture of the globe. with consequent infection of the operative field. Therefore, about three years ago, I had Pilling & Company make me a special snare that serves so admirably that I feel that I should pass it on to others.

The instrument is a modification of the Lewis snare with the shank made smaller, flattened at the end, and curved just enough to reach the nerve without stretching it. It is slotted on the sides, so as to receive the wire, and the stem is long enough to draw in the wires for cutting the nerve and is perforated at the end just opposite the slots in the cannula. It takes a number seven piano wire nine inches long and is easily threaded if the ends of wire are bent slightly before inserting. Of course, the

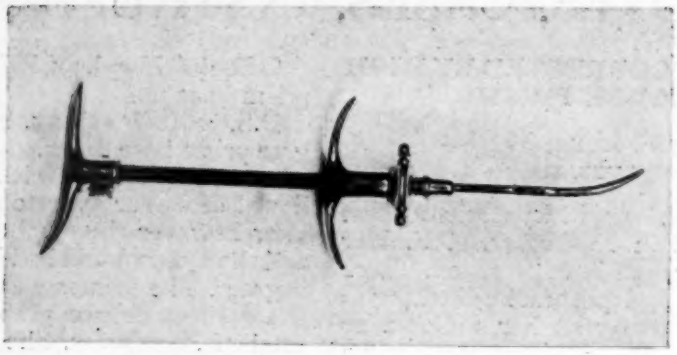


Fig. 1. Enucleation Snare (Banner).

conjunctiva, muscles and capsule, must be dissected the same as in all these operations. The advantages of the instrument are:

1. It makes a clean cut of all the tissues with the least possible amount of traumatism.

2. Hemorrhage is reduced to a minimum.

3. It cuts the nerve just back of the eyeball, and leaves the stump neither too long nor too short, closing the end of the retinal vessels.

4. It shortens the duration of the operation and lessens the possibility of infecting the wound.

5. It leaves the eyeball intact, making a better specimen for postoperative examination.

Greensboro, N.C.

SOCIETY PROCEEDINGS

Edited by Dr. H. Rommel Hildreth

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

February 16, 1931

Dr. Hilliard Wood presiding

Gun shot wound of the orbit

Dr. Robert Warner reported the following case; M. K., aged five years was first seen on January 9, 1931. The history was, that on January 4, he was hit in the right eye with B.B. shot. He was immediately seen by his local oculist who applied antiseptics and boric acid compresses to his eye. The shot was localized by x-ray as being within the orbit to the temporal side of the eye ball. The wound of entrance was through the outer portion of the upper lid. An unsuccessful attempt was made to remove the shot. When seen by Dr. Warner five days after the accident there was much swelling of the upper lid with a large infected wound extending through the outer portion. The swelling was so great that the eye ball could not be seen. The patient was sent to the hospital where the wound was washed out with mercurochrome and iced boric acid compresses were applied to the lid every two hours. The following day the swelling had diminished to the extent that the eye ball could be seen. The cornea appeared normal; iritis was present and vision was light perception. Atropine was instilled and the mercurochrome and boric acid treatment continued.

On January 14, while dressing the wound the shot appeared at the surface and was easily removed with forceps. By this time there was no pus in the wound and very little swelling present. The patient stated that he could see well out of this eye, and ophthalmoscopic examination showed a normal fundus. The iritis cleared and the patient made an uneventful recovery.

Torpedo injury of the eye

Dr. W. G. Kennon reported a case in which the patient was struck in the left eye by a portion of an exploding torpedo.

On examination there was found a small laceration of the skin near the margin of the upper lid. On turning the lid an oblique fracture through the tarsal-plate was seen. There was no injury to the bulbar conjunctiva, and the cornea was normal. The tension on palpation was normal. The anterior chamber was deep and about half full of blood. A few small blood clots were seen on the upper portion of the iris. The pupil was partly dilated and inactive. Vision of injured eye was hand movements. Ophthalmoscopic examination was not possible.

Right eye vision was 20/20 and the eye was apparently normal in every respect.

One percent atropine was instilled in the left eye and the patient instructed to go home and be quiet.

The following day he began having severe pain in the eye. When seen on the succeeding day the anterior chamber was found to be entirely filled with blood and the tension of the eye was distinctly elevated, as determined by palpation. The patient had not obeyed advice to remain quiet.

Pilocarpine and eserine were used. The next day, the patient was more comfortable but the tension of the eye was unchanged. After 24 hours, the eye looked decidedly better, and there was apparently some absorption of the hyphemia. The condition showed little or no improvement during the next week.

Two weeks after the accident severe pain recurred. The cornea took on a greenish hue, the upper lid became slightly swollen, there was congestion and a ciliary staphyloma developed in the lower quadrant. Enucleation was

performed eighteen days following the accident.

The entire interior of the eye was filled with blood. There was no foreign body.

The unusual features about this injury were a definite fracture of the tarsal plate and what appeared at first to be a minor injury, developed into a condition so serious as to destroy the eye.

H. C. SMITH,
Secretary.

ROYAL SOCIETY OF MEDICINE, LONDON

Section of Ophthalmology

March 13, 1931

MR. ELMORE BREWERTON, president

Tumor of the iris

MR. LESLIE PATON showed a man first seen ten days ago. The condition seemed too dangerous to leave alone, and as the eye had 6/5 vision the exhibitor was very unwilling to sacrifice the eye. He regarded the condition as sarcoma and had determined to try the effect of radium. Most members who saw it thought it was not inflammatory.

Discussion. MR. BREWERTON said he felt satisfied that it was a new growth, and if radium should fail the question was whether iridectomy should be done to settle the diagnosis.

Glioma of the retina

MR. R. FOSTER MOORE showed a boy who had had the left eye removed because of glioma when five months old. The lad's brother had had glioma and both eyes were removed, but in spite of the treatment the boy died. In the present case he put a radon seed into the growth, and the latter then started to disappear. In January the condition was stationary. A second seed was inserted in April, 1930 which was lost in the eyeball, but was eventually recovered and fixed in position, no damage having been done. There was now certainly a recurrence.

Proptosis

MR. R. A. GREEVES showed a case in which the condition was much the same in 1925 as at the present time. There was fairly good vision. The conjunctival vessels were injected. He thought it was hemangioma of the orbit.

Krukenberg's spindle

MR. R. A. GREEVES presented a case in which the oval line of pigment was in the posterior layers of the cornea. Sometimes the pigmentation was congenital in this disease; it was so in this case. It could be acquired, and he had seen it develop in a woman aged 62 years.

Retinal moles

MR. R. A. GREEVES showed a patient with this condition. Though not of great clinical importance they were not very common. He thought these were not malignant, but were melanomata of the pigment epithelium.

Familial macular degeneration

MR. R. A. GREEVES showed two patients, the sight of one, aged 18 years was good until her tenth year, when it began to fail. Now it was 3/60 in each eye. The other was aged 11 years and had good sight until 18 months ago. Now the vision was 6/36 in each eye.

Cataract after electric flash

MR. M. H. WHITING showed a man, aged 46 years who had been seen three days after the flash. There was considerable spasm and colorphobia. The cornea did not seem to have been much injured. The vision had now improved from O.D. 6/12 and O.S. 6/9.

Cupped discs and binasal hemianopsia

MR. M. H. WHITING showed a man who had been treated for syphilitic meningitis. The cupping did not extend over the whole area of the disc. There was a ringed scotoma which merged into the nasal defect. The hemianopsia might have been due to unusual glaucoma and he believed the affection of the fields was due to thickened carotid arteries, or to thickened meninges pressing on the side of the optic chiasma.

Iridocyclitis

MR. WHITING next showed a 16 year old boy who had palpable supraclavicular glands, and in whom the carotid arteries seemed to be enlarged. The Wassermann was negative and the blood count normal. A lymph gland removed from the right side of the neck was typically tuberculous. The mediastinal glands seemed to be enlarged.

Discussion. MR. PENMAN suggested ultra-violet treatment.

Sclerosing keratitis

MR. COLE MARSHALL showed a case in which the sclerosed mass was practically in the center of the cornea, and very deep vessels had entered into it, as revealed by the slit-lamp.

Discussion. MR. BREWERTON described a similar case in a boy, aged 19 years, in only one eye. There seemed to be much pus in the substance of the cornea. At operation he found the mass to be between two layers of the cornea. After scraping there was practically complete recovery.

MR. O. G. MORGAN thought this case did not quite correspond with the classical description of sclerosing keratitis and he did not think an operation would show any cavity.

Retinal detachment

MR. H. NEAME showed a case in which at one time he lost hope and he had failed in his efforts to bring about replacement of the retina. The patient's attitude was not encouraging. The successful result now seen showed what patience and perseverance would accomplish.

(Reported by H. Dickinson)

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

March 16, 1931

DR. HILLIARD WOOD presiding

Tularemia of oculoglandular type

DR. J. L. BRYAN reported that on December 16, 1930, Mrs. W. L. N., aged thirty years, came to him complaining of swelling and soreness of left eye, also swelling in front of the left ear and under the jaw. This began two weeks previously with pain, inflammation and discharge from the eye. A nasal mucopurulent discharge with frontal and temporal headache also existed. Four days before this onset the patient had skinned two rabbits.

Examination showed the left lids swollen and red. A mucopurulent secretion glued the lashes together. In the extremely inflamed conjunctiva of both lids were numerous ulcers with yellow necrotic centers and water-like rims. There was one nodule at three o'clock, 3 mm. from the limbus on the globe. The cornea was clear, there was no iritis and vision was normal.

The preauricular and submaxillary glands were swollen, the temperature 100 3/5°, pulse 94 and there was slight leukocytosis. The ears and nose were normal but the throat showed numerous superficial indurated ulcers about the tonsils and posterior pharyngeal wall. On the face, neck and shoulders, arms, chest and back was a discrete eruption. The blood Wassermann was negative.

In the hospital in addition to fluids and purging the eye was irrigated four times a day followed by two percent zinc sulphate. Applications of two-tenths percent sodium recinoleate were made to the throat night and morning and a spray of detoxol every two hours.

In two months the preauricular glands had softened and following incision a small amount of pus was obtained. The diagnosis was confirmed by the agglutination test.

Discussion. DR. ROBERT WARNER had read where one attack of tularemia produced immunity.

DR. J. L. BRYAN in closing said that he did not think zinc sulphate was a specific, but that he treated the patient with it and there had been improvement. All of the ulcers were alike, but the ulcers in the pharynx were not so deep. He thought the picture of the tularemic condition was different from anything he had ever seen, and was in no way like Paranaud's conjunctivitis.

H. C. SMITH,
Secretary.

CHICAGO OPHTHALMOLOGICAL SOCIETY

March 16, 1931

DR. HARRY S. GRADLE, president

Restoration of socket

DR. P. D. O'CONNOR presented a woman who had lost an eye from lye burn when an infant. The socket was completely obliterated. Inasmuch as she found it difficult to get employment on account of this disfigurement, it was decided to attempt to restore the socket by an epidermal graft as described by Dr. John M. Wheeler in the American Journal of Ophthalmology in 1921. The conjunctiva was incised and the dissection was made with scissors parallel to the tarsus and was carried to the orbital margin inferiorly and to the sides. An external canthotomy was done. A mould of dental compound was formed and around this was placed the epidermal graft from the thigh. This was inserted in the socket, and a pressure bandage was applied. After five days the dressing was changed. The mould was removed after three weeks. Following this, it was possible for the patient to wear a glass eye. The socket had not contracted in the eight months since the operation was performed.

Discussion. DR. SANFORD GIFFORD emphasized one essential point in keeping the graft from shrinking. It must adhere to the orbital margin above and below; if not it would shrink after the mould was removed. He had seen one case in which shrinkage had gradually occurred because it was not adherent.

Retinal tuberculous vasculitis and hemorrhage

DR. GEORGE F. SUKER presented a case which had been seen by Dr. Weiner some time ago. Conditions had changed in the left eye since, but the same diagnosis was now made. There was arrested tuberculosis; the x-ray showed the peribronchial glands still enlarged as well as some calcareous deposits in the lungs. There was almost complete detachment of the retina in the right eye; vision was practically normal in left eye. The case was interesting with reference to one shown by a member at the previous meeting as a massive vitreous hemorrhage on a luetic basis. Syphilis was not prone to cause vitreous hemorrhages in young persons as in older ones. It did not occur unless there was an endarteritis. The prognosis of the hemorrhage on a tuberculous basis was relatively good, while that of luetic origin was not. There was no reason why this man should not maintain good vision in the left eye. It was interesting to note the formation of anastomoses in the outer superior temporal region of retinal vessels in the area of hemorrhage.

Pineal gland calcification with hemorrhage

DR. GEORGE F. SUKER presented a patient who noted inability to see toward the left after a cardiovascular lesion. There was a complete left homonymous hemianopsia with sparing of the maculae so that central vision was normal. X-ray of the pineal gland showed a little lateral displacement. The gland was large, calcareous and had a distinct shadow extending downward on to the corpora quadrigemina. There evidently was a hemorrhage in and around the gland. All serological tests were negative. The patient stated that vision had not changed since the start of his trouble. In a case of pineal gland tumor shown some time ago, in which there was a central scotoma, x-ray, pituitary extract, potassium iodide and mercury rubs did much to restore practically normal vision. These tumors were not very common. Pineal gland disturbances usually appeared in men of about fifty to sixty years of age, and in women about the age of menopause.

Mooren's ulcer

DR. SANFORD GIFFORD showed a patient, who, in his opinion had had a Mooren's ulcer which was now healed, although it had been stated that if the ulcer became well it was not a rodent ulcer. Axenfeld's textbook showed a picture of a very advanced Mooren's ulcer, which was characterized by the fact that it started at one point near the limbus, and there was marked

hypertrophy at the limbus. It healed as it advanced across the cornea. It spread around the ends and then into the middle. In this case the ulcer went around and extended upward; there was a large area of bulbar conjunctiva which was raised, thickened and red. One could not be sure of the diagnosis in the early stages.

In treating this case, several places were touched with iodine, and intravenous protein was administered, but nothing checked the course. Therefore a delimiting keratotomy was done, about 2 mm. long in front of what seemed to be the most active point of ulceration. Cocain was injected subconjunctivally, and every day the lips of the wound were touched with a curved cystotome and the aqueous allowed to escape, which relieved the tension from the cornea. After being kept open a week or so it began to heal. These ulcers were presumed to have some connection with malnutrition and this patient was given codliver oil, which may have helped.

Discussion. DR. T. D. ALLEN said that Saemisch did not keep the wound open in these cases. He asked whether there was ingrowth of epithelium of the cornea, or whether there was danger, in a shallow anterior chamber, of the epithelium growing into the anterior chamber.

DR. SANFORD GIFFORD had never seen any cysts of the anterior chamber from ingrowing epithelium after keratotomy. In reply to Dr. Von der Heydt, he had seen cases in serpent ulcer in which the cut was not made the whole length and the ulcer progressed around the cut. It was supposed that since the epithelium resisted the ulcer longer than the corneal stroma it might act as a barrier. The reason the cut was made in the cornea instead of at the limbus was so that the incision might be kept open each day.

Bilateral lacerations of the cornea

DR. LENNON showed a patient who was injured in an automobile accident in December, 1930. It was difficult to ascertain the vision on account of the lacerations of both corneas; the approximate vision was 10/200 each eye. In the left eye the lacerations extended from three to nine o'clock on the limbus and also involved the sclera 2 mm. further, extending through the ciliary body. The right cornea was lacerated, the linear lacerations being about 5 mm. long. There was iris prolapse in this eye. Dr. Gifford saw the case, and before definitely deciding upon enucleation, a flap operation was done on both eyes. The flap was brought down from above in the left eye, and a sliding flap was made in the right eye. A traumatic cataract developed. The patient left the hospital in about a month, and returned one month later. A keratome incision was made and the lens remains removed. With correction, vision was 10/10, O.D., 10/200, O.S. It was hoped to increase the vision later by doing an iridectomy in the left eye.

Discussion. DR. SANFORD GIFFORD said that in this case apparently the lens was not injured by the cut clear across the cornea. The only reason for trying to save this eye was because the other eye was injured and might be lost through the result of cataract operation. The patient was seen just after the injury and it was thought not dangerous to do this and wait a day to see if infection developed. Since it did not, it seemed safe to keep the eye.

DR. ROBERT VON DER HEYDT suggested that the eye be left alone. There was normal vision in the one eye and 10/200 in the other. Further interference with the ciliary body or iris was not indicated, nor was it safe.

Chiasmal symptoms in intracranial tumors

DR. LOYAL DAVIS said a study was made of the cases of intracranial tumors in which chiasmal symptoms had been the outstanding if not the only symptoms of intracranial pathology. Examples were given of various syndromes which pointed to diagnostic entities. The surgical treatment of tumors in such a location, as well as their pathological characteristics, was considered.

Discussion. DR. PERCIVAL BAILEY said that from the standpoint of the neuro-

logic surgeon, this was a most complete presentation. The symptoms described were in line with those worked out by Dr. Harvey Cushing. With respect to gliomas of the optic chiasm, a preoperative diagnosis might often be made by taking x-rays of the optic foramen, which could easily be done by directing the rays along the wall parallel to the orbit. Of course gliomas of the optic chiasm were not favorable from the operative standpoint, but could be treated by x-ray therapy, which spared the surgeon the back-breaking task of opening the skull merely for the doubtful pleasure of closing it again. Very commonly the patient had von Recklinghausen's disease. There were also symptoms which had to do with the fundus alone whereby the diagnosis had been made by ophthalmologists, but this was among the refinements of ophthalmology.

A study of the relation of the basal metabolic rate of progressive myopia

DR. LOUIS BOTHMAN read a paper on this subject published on p. 918 of this issue of the American Journal of Ophthalmology.

Discussion. DR. MEYER WIENER, of St. Louis, said that about four years ago when he had made a preliminary report on the use of epinephrin in myopia, Dr. Calhoun had told him that he was making some experiments on the basal rate in progressive myopia, which work had apparently not been published. The interesting point in Dr. Bothman's work was the taking the basal metabolic rates in hyperopic patients as well as in myopic, with the result that only one case of a low rate was found in the hyperopic cases, and just the opposite in the myopic, one case of raised basal metabolism. That looked almost conclusive. There was apparently no one cause for progressive myopia. The fact that myopia had been treated with epinephrin was no reason for stating that all cases were due to lack of epinephrin. His reason for using it in progressive myopia was because of the increase in metabolism in the tissues produced by its use. This had been proven not only in the sclera but in practically all tissues. Dr. Ince found most cases to be of the asthenic type; his own observations were the opposite. His patients were usually stout and heavy, inclined to be rather slow and lazy mentally. One would suspect hypothyroidism in such cases. Little was known of the action of the ductless glands. There was a sort of cooperation between the various glands; no particular one could be said to be the cause of any of these diseases. It might be a sort of working together, and perhaps the thyroid might be the one in certain types of progressive myopia. It was a fact that administration of thyroid extract increased the function of the adrenals.

Gliomas of the retina; including the results of studies with silver impregnations

DR. ROY GRINKER read a paper on this subject.

Discussion. DR. RAMON CASTROVIEJO said that when in Spain recently he had the opportunity of seeing slides made by Dr. Ascunce, who, in 1905 gave a beautiful description of every type of glioma cell stained by the Golgi-Cajal method. Later Dr. Munoz-Urra, in 1923, gave the best description of the elements which constituted glioma, using for staining the method of Rio-Ortega. His description confirmed the opinion of Dr. Ascunce. In 1927 Dr. Ascunce stained other eyes by the Rio-Ortega method, and had obtained the same type of cells as Munoz-Urra described in 1923, which description had been accepted as the model in German literature.

ROBERT VON DER HEYDT,
Secretary.

AMERICAN JOURNAL OF OPHTHALMOLOGY

PUBLISHED MONTHLY BY THE OPHTHALMIC PUBLISHING COMPANY

EDITORIAL STAFF

LAWRENCE T. POST, editor
524 Metropolitan building, Saint Louis
WILLIAM H. CRISP, consulting editor
530 Metropolitan building, Denver
EDWARD JACKSON, consulting editor
217 Imperial building, Denver
CLARENCE LOEB, associate editor
1054 Tower Road, Hubbard Woods, Illinois
HANS BARKAN
490 Post street, San Francisco
EDWARD C. ELLETT
Exchange building, Memphis, Tennessee
HARRY S. GRADLE
58 East Washington street, Chicago
M. URIBE TRONCOSO
515 West End avenue, New York
JOHN M. WHEELER
30 West Fifty-ninth street, New York

Address original papers, other scientific communications including correspondence, also **books for review** and reports of **society proceedings** to **Dr. Lawrence T. Post, 524 Metropolitan building, Saint Louis.**

Exchange copies of medical journals should be sent to **Dr. William H. Crisp, 530 Metropolitan building, Denver.**

Subscriptions, applications for single copies, notices of change of address, and communications with reference to advertising should be addressed to the **manager of subscriptions and advertising, 508 Metropolitan building, Saint Louis.** Copy of advertisements must be sent to the manager by the fifteenth of the month preceding its appearance.

Authors' proofs should be corrected and returned within forty-eight hours to the **editor.** Twenty-five **reprints** of each article will be supplied to the author without charge. Additional reprints may be obtained from the printer, the George Banta Publishing Company, 450-458 Ahnaip street, Menasha, Wisconsin, if ordered at the time proofs are returned. But reprints to contain colored plates must be ordered when the article is accepted.

THE OXFORD OPHTHALMOLOGICAL CONGRESS

Around certain organizations there is often flung, in the course of time, a sort of romantic halo, associated partly with the place of meeting, partly with the passage of the years, and partly with the personalities of those who have had an important share in organization or proceedings. Such a halo clings in Germany to the Heidelberg Congress, and in the United States to the American Ophthalmological Society.

In the British Isles, one of the younger medical organizations, the Oxford Ophthalmological Congress, has rapidly acquired a mellowness of dignity and respect such as usually belongs only to much older bodies. The credit for this position may perhaps be attributed to several factors, including the university setting and the modern educational movement in ophthalmology to which the Congress was incidental in the purpose of its founder, Mr. Robert Walter Doyne, first Reader in Ophthalmology at the University of Oxford.

The position accorded to the Congress does not of course in any way belittle the worth or importance of the Ophthalmological Society of the United Kingdom, in whose annual volume of transactions the proceedings of the Congress are published. On the other hand, certain political and other considerations, especially perhaps the complications arising from the national health insurance laws, seem to have prevented such cordial coöperation between special eye organizations and the eye section of the British Medical Association as exists on this side of the ocean between the American Ophthalmological Society, the American Academy of Ophthalmology and Otolaryngology, and the Section on Ophthalmology of the American Medical Association.

The social aspect of medical assemblies has an unmistakable significance and is capable of contributing largely

to scientific and professional progress; and this aspect has played an important part in the career of the Oxford Ophthalmological Congress. The social activities of the Congress are carried on in Keble College, a young institution quite in accord with the educational, architectural, and other traditions of Oxford. Many of those attending the Congress are housed in the students' chambers, rendered available for this purpose by the summer vacation. Meals, including the formal annual banquet, are taken in the hall of the college, where one is surrounded by portraits of ancient and modern worthies of the educational and ecclesiastical world. The Junior Common Room of the College serves as a sort of lounge and writing room for members of the Congress. The scientific proceedings of the Congress are conducted in the department of human anatomy of the museum of the University of Oxford, across the street from Keble College.

This year saw the twenty-first annual meeting. At the founding of the Congress by Mr. Doyne, in 1910, the membership was about one hundred and fifty. It is now over four hundred, and the annual attendance has reached two hundred. Some well-known American ophthalmologists are members of the Congress, and this year's Congress included papers by several American members, as well as special contributions by distinguished foreign guests, Bailliart of Paris, Gjessing of Drammen (Norway), and Schüller of Vienna.

The scientific program was well arranged and included a large amount of important and interesting material. The first morning was entirely occupied by a symposium on the diagnosis of intracranial newgrowths, which was ably introduced by Mr. Leslie Paton, Dr. Traquair, Mr. Norman Dott, and Professor Arthur Schüller. The present writer, while recognizing the value of symposia, is not always sufficiently grateful for the manner of their presentation. Too often the amount of time occupied by individual speakers is excessive, while the details are not always well balanced or attractively arranged. It would be an advantage, to listeners at least, if a definite limit of time might always be assigned to each speaker and more or less definitely insisted upon by the chairman. It would be a further advantage if all those placed upon a symposial program could confer beforehand as to the fields to be covered and as to the manner of presentation. But, in spite of some possible exception along these lines, the symposium as to intracranial growths was impressive and valuable. It will of course be published later, with the other proceedings of the Congress, in the annual volume of the Ophthalmological Society of the United Kingdom.

The afternoon address by Professor G. Elliot-Smith on the "Evolution of the Instruments of Vision" was finely delivered, with all the interest and authority attaching to everything said or published by this eminent authority on anthropology and evolution; the pleasure and satisfaction with which it was received being perhaps rather enhanced by the fact that most of the ideas presented were already somewhat familiar to those who had heard or read Professor Elliot-Smith's earlier addresses. Professor Elliot-Smith has recently been deeply absorbed in a study of the new type of early fossil man discovered near Peiping.

Mr. Bernard Cridland, Master of the Congress, was unfortunately prevented by illness from attending most of the meetings but was joyfully welcomed when he turned up on the last day. His place had been taken by Mr. Philip H. Adams, Deputy Master, who also delivered the Doyne Memorial Address. Mr. Cyril Walker was elected Master for the ensuing year; and Mr. Russ Wood, the very efficient and genial Secretary, was reëlected. *W. H. Crisp.*

PROBLEMS FOR THE OPHTHALMOLOGIST

The progress of medicine has always been beset by difficulties and perhaps never more so than today. Obviously it is possible to consider only one or two of these in a short article and even these from only a few viewpoints.

The outstanding problem of any

science is its advancement as a science, which depends on the discovery of new facts, methods and ideas. In medicine some of these will originate in the laboratory and some will be derived from the study of disease in the patient, so both laboratory worker and clinician will have a share in contributing to this progress. The opportunity of the former has been enhanced by better facilities for research and more financial backing. The public is awakening to the expediency of encouraging research in all lines and has become increasingly generous in contributions for this. The improved conditions are attracting each year more workers of high caliber into these fields. There can be no doubt that advancement along these lines will continue at a rapid pace. For the clinician too the path has been made easier by the increased facilities for study of his cases. He has better instruments of precision and far more help from laboratory and consulting technicians than his predecessors had so that practically any study which he desires may be carried out on the patient.

The situation, for the ophthalmologist, as regards service to the patient is far less satisfactory. Patients who have eye lesions indirectly related to disease elsewhere in the body constitute a fairly large and certainly a very important and interesting group. The method of earlier years was to study the affected eye carefully, obtain as accurate a history as possible and make such general examination as the ophthalmologist himself was capable. With this data alone by which to be guided he treated the patient. The next forward step was the referring of the patient to an internist who had equipment for making a much more complete study than the oculist. Soon the internist called to his aid many special tests such as blood, dental, x-ray, otolaryngological, genitourinary and tubercular examinations. When all these can be made the chances of an accurate diagnosis, hence rational and effective therapy are greatly increased. In fact to do less than submit the patient to these examinations now-a-days is not giving him the full benefit of modern science.

Unfortunately to do this is not always easy. The difficulties confronting doctor and patient may be many but especially hampering are lack of local facilities and lack of funds. The former affects everyone though the wealthy may be able to overcome it by travel to a place where these tests can be carried out.

What can be done to overcome this difficulty? One answer is the construction of adequate hospitals within reach of all. Whether this should be done by private enterprise or otherwise may depend on the community. This development can be only gradual and many problems will have to be solved and adjustments made.

For the patients of average means, earning from fifteen hundred to two thousand dollars a year, who constitute by far the biggest group in our country, the problem of financing these examinations is a very serious and often an unsolvable one unless these individuals consider themselves fit subjects for charity and seek help on this basis. This would be a most pernicious outcome. It would degrade the patient, be an unjustifiable burden on others and deprive the physician of a remuneration to which he is entitled. The practice of institutions of hospitalizing these patients on a charity basis is wrong.

Hospital staffs should establish a systematic routine for the study of these patients, especially with a view to the unearthing, if possible, of foci of infection and require a fee commensurate with the patient's means. Here as in everything else the cost can be greatly reduced if a large enough number are handled. To decide on a standard routine for the discovery of infective foci should not be impossible and it would be an immeasurable service and most welcome to the ophthalmologist to be able to send a patient to such a center provided that the profession was protected by an understanding that no patient would be admitted except on the recommendation of the patient's physician. Furthermore the physician would be entitled to a fee in every case except those in which nothing was paid to the hospital. If the patient can pay

nothing to the hospital, then and then only, should the patient pay nothing to the doctor. If the patient can pay something to the hospital he can pay something to the physician.

By such a service as this the average wage earner could receive the very best at a cost within his means whereas if he is referred to half a dozen specialists independently he could be asked to pay far more than his income would justify. The danger of these institutions is their abuse. It is human nature to want to get something for nothing. If to this desire of the patient is added the inclination of an organization, for many reasons, to take care of as many cases as feasible, the temptation to adopt the attitude of "come one, come all" is apt to be overwhelming and between the two, the doctor and the taxpayer and the contributor to charity suffer.

Another unsolved problem is that of testing refraction and supplying glasses to these patients of the middle wage class at fees which they can pay. Ophthalmologists want to take care of these people and most certainly should do so but some more economical method of doing this than is current in most places will have to be devised or testing the refraction of the average wage earner will pass out of the hands of eye physicians. Much has been written on this complex subject and space will not permit of discussion at this time but it behooves our national and local ophthalmic bodies to be giving this major problem their earnest consideration. It is not a problem for the individual only but for the entire profession.

Lawrence T. Post.

BOOK NOTICES

Bulletins et Mémoires de la Société Française d'Ophtalmologie, 1930. Paper, octavo, 516 pages, with numerous illustrations in black and white. Masson et Cie, Paris.

This volume constitutes the 1930 report of the French Ophthalmological Society whose annual meeting was held May 12-15 in Paris. In it are included 63 papers presented before the Society, with full discussion. Among these are "Etiology and treatment of glaucoma secondary to extraction of the lens" by V. Morax, "Researches on the dilator of the pupil" by M. E. Redslob, "The effect of experimental compression of the globe on the visual acuity" by P. Bailliart, "Decompressive trephine of the optic canal" by Worms, "Sclero-retinal thermo-cauterization in detachment of the retina" by Van Lint, "Hereditary degeneration of the cornea of the Groenouw type" by F. E. Koby, "Condition of the vitreous after cataract extraction" by de Saint Martin, and "Tarsal biomicroscopy in conjunctivitis" by Cuènod and Natof. A short summary of each article will appear in the abstract department of this journal.

The report of Professors Duverger and Velter entitled "Biomicroscopy of the lens in its normal and pathologic states", submitted to the Society May 13, 1930, is published as a separate volume and will be reviewed separately. It constitutes the third section of an atlas of ocular biomicroscopy whose first two volumes "Microscopic examination of affections of the cornea by means of the slit lamp" by Gallemaerts, and "Biomicroscopy of the anterior chamber, the iris, and the ciliary body" by Marvas, have already appeared. These are for sale by Masson & Cie, 120, boulevard Saint-Germain, Paris.

The report for 1931 entitled "Heredity in ocular disease" will be given by Marnix Van Duyse, while the report for 1932 entitled "Biomicroscopy of the vitreous body in the normal and pathologic states" will be given by Koby and Redslob. *Phillips Thygeson.*

Seeing—a Partnership of Lighting and Vision, M. Luckiesh and Frank K. Moss, 241 pages, numerous illustrations, charts and tables. Price $5.00. The Williams and Wilkins Company, Baltimore, 1931.

When one stands thoughtfully between the physiological facts of vision, on the one hand, and the physical facts which practically go to make visibility, on the other, he finds himself in a field which at first presents no unusual scientific complexity. That is, not until

he makes the attempt at quantitative application of the facts obtained in the laboratory under simplified conditions to any of the million different situations in which we human beings of all walks of life are calling light into partnership with our eyesight; situations, each of which is indefinitely complex and more or less impossible of exact description. If these sentences have failed to make their point clear, it is owing to reasons therein implied, as well as to lack of space. The alternative is for the reader to read the book, for nowhere else will he find proposed such a wide variety of problems which stand between lighting and vision.

It is perhaps superfluous to say at this point that the book is suggestive rather than conclusive. Possibly, also, this would be misleading; for certain definite trends have emerged from the scientific study of lighting and vision, and certain indications as to where these may be applied, and where they may not.

As to the ostensibly exact statements contained in the book, however, one who is familiar with the material will have doubts as to the value of some of the plotted curves, or of the numerical data given in some of the tables. These range in content all the way from accurate reports of experiment to tables or diagrams representing generalizations of unstated origin. Table VI, for example, occupying a large part of page 57, is a set of computations based on an admitted assumption which is wholly untrue of many of the proposed hypothetical situations.

On the whole, the book leaves one with the feeling that the material might have been much better arranged. The documentation of the original work about which the discussion turns might be more complete. In one place, at least, even quotation marks are omitted, let alone credit given to the author. Of forty-eight titles in the bibliography there are thirty that are indebted for their authorship to one or both of the authors of the book, and five more to others at Nela Park; the literature from outside has been scantily treated. *Percy W. Cobb.*

Recent advances in microscopy. By A. Piney, M.D., Director of Pathological Department, Cancer Hospital, London, 260 pp., 83 illustrations. Philadelphia, P. Blakiston's Son and Company, Inc., 1931.

There are four principal subdivisions of this book. The first, devoted to the medical sciences, occupying thirty pages, includes the microscopy of the body in general and is by Piney. The second chapter on the microscopy of the living eye is by Basil Graves. It is entirely given over to slit lamp studies. It is a condensed résumé of the present knowledge, much of the work being that of the author as previously reported in various journals. The illustrations are for the most part diagramatic. For the scientist, other than the ophthalmologist familiar with the slit lamp, the chapter is of interest and value. It is too elementary for the ophthalmologist familiar with the slit lamp and not sufficiently comprehensive as a text for the ophthalmologist desiring to learn about the slit lamp. The third chapter contains 79 pages concerning zoölogy and the fourth 86 pages about botany. *Lawrence T. Post.*

ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is only mentioned in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history

---

9. CRYSTALLINE LENS

Knapp, Arnold. **Extraction of cataract.** Arch. of Ophth., 1931, v. 5, April, pp. 575-880.

In this paper, the third hundred successive extractions of cataract in the capsule following preliminary subluxations with the capsule forceps are reported. The author predicates that cases suitable for this method must have a capsule which is dense enough to hold the grasp of the forceps. For this reason it is not attempted in patients under fifty years of age. The capsules of cataracts with a glistening cortex usually rupture. The sclerosis type with posterior cortical opacity dislocates readily. The same is true of those with a thickened capsule. In Morgagnian cataract, the capsule is difficult to grasp. The section must be large, and, therefore, almost entirely scleral. Kalt forceps, with blunt tips like a bisected cup, should be used. They should be applied to the lower portion of the capsule. During their manipulation, the Smith hook makes counterpressure at the lower limbus backward and downward. When subluxation takes place, the lower margin of the lens appears in the pupil and the forceps are removed. In these 100 cases, 93 tumbled. In 5 of these the capsule ruptured. There was head presentation in 3 cases, and in 4 the capsule was grasped a second time and pulled out. Slight vitreous loss was experienced in 7 cases. Iridocyclitis was present in 1 case. The wound reopened in 10 cases. There was expulsive hemorrhage in 1 case. The retina was detached in 2 cases. Paralysis of the iris occurred in 1 case. Glaucoma was not observed. In 56 cases the vision was 20/20, in 18 it was 20/30. *M. H. Post.*

Poos, Fr. **Clinical observations on Soemmering's crystalline swelling in myopic eyes operated upon according to Fukala.** Klin. M. f. Augenh., 1931, v. 86, April, p. 449.

In two men, aged forty-seven and forty years, whose lenses had been extracted according to the method of Fukala, the former twenty-five and the latter thirty years previously, on account of high myopia, a crystalline swelling had developed. This swelling had been dislocated by trauma, and when the patient stooped it floated into the pupillary area with impairment of vision. The two cases are illustrated. From these and a similar case of Wessely's the author concludes that the predisposing factors in dislocation of Soemmering's crystalline swelling are trauma, loss of the lens at a youthful age, and progressive myopia with subsequent gradual degeneration of the vitreous. *C. Zimmermann.*

Salit, Peter W. **Biochemical investigation of the nitrogen weight and water content of crystalline lenses.** Arch. of Ophth., 1931, v. 5, April, pp. 623-633.

The water content of the lenses of animals decreases with age. In cattle there is a rather rapid, uniform decrease in this constituent from five weeks to two years of age. It has been suggested that this dehydration may be somewhat responsible for the formation of cataract. The total solids, chiefly proteins, naturally show an increase. Senile human cataractous lenses, on the contrary, show a decrease in protein content. So far, no experimental evidence is available to explain this seeming inconsistency. The total nitrogen of the lens increases from 4.14 percent in calves of two months, to 5.79 percent in cattle of two years. The nonprotein nitrogen of cattle lenses averages up to 100 mg. per 100 grams. In rabbits it is about 5 percent less. The protein increases from 25.86 percent in calves of two months to 35.71 percent in cattle of two years. The lens of the adult rabbit contains 33.4 percent, that of the adult sheep 23.96 percent. The water content in cattle decreases from 68.6 percent in calves of five weeks to 64.6 percent at two years. The adult rabbit lenses give an average of 59.2 percent. *M. H. Post.*

10. RETINA AND VITREOUS

Bidault, R. **Retinal tonometry in cases of arterial hypertension.** Ann., d'Ocul., 1931, v. 168, April, pp. 255-264.

The tension of the central retinal artery is compared with that of the general circulation in cases of hypertension. The author finds that hypertension of the retinal artery is usually present in a variable degree in these cases. *H. Rommel Hildreth.*

Cowan, Alfred, and McAndrews, L. F. **Retinal detachment: a method of accurately localizing tears.** Arch. of Ophth., 1931, v. 5, May, pp. 760-765.

After reviewing the usual methods, the authors suggest a means used by themselves some years ago, whereby the location of the tear is plotted on a tangent screen. In this method they assume the inside diameter of the globe to be 22 mm., under which conditions each degree in the field is equal to 0.25 mm. on the retina, on which base the perimetric chart is lined with squares whose sides would correspond to one mm. in the retina. The patient is placed one-half meter from the tangent screen, the affected eye opposite the fixation point. He is then told to move his eye along the field as far as possible towards the side involved by the tear. The examiner then locates the tear with the ophthalmoscope, which line of direction is projected back on to the tangent screen by an assistant. This line of direction must be corrected by a decrease of one-fourth in extent, so that if the patient has rotated the eye 50 degrees and the examiner's angle is 40 degrees, the lesion lies 80 degrees outward in the horizontal meridian. Should the examiner's angle be 20 degrees above the horizontal, this also must be reduced in a similar manner to 15 degrees; the lesion, therefore, is located and charted as being 80 degrees from the center and on the 15 degree meridian. Ninety degrees on the chart corresponds to the ora serrata, lying 8 mm. behind the lens. Our lesion will lie, however, 3.5 mm. inward from the 90 degree meridian, or 8 mm. plus 3.5 mm., equaling 11.5 mm. behind the lens and 5 mm. above this point. *M. H. Post.*

Ellett, E. C. **Retinal detachment.** Arch. of Ophth., 1931, v. 5, May, pp. 784-788.

This paper contains a review of the work done by Gonin, Sourdille, Finley, and Badeau on retinal detachment. *M. H. Post.*

Goldstein, I., and Wexler, D. **Niemann-Pick's disease with cherry-red spots in the macula; ocular pathology.** Arch. of Ophth., 1931, v. 5, May, pp. 704-716. (See Section 17, Systemic diseases and parasites.)

Goldstein, I., and Wexler, D. **Rosette formation in the eyes of irradiated human embryos.** Arch. of Ophth., 1931, April, p. 591. (See Section 15, Tumors.)

Gonin, J. **Thermocautery obliteration of tears in detachment of the retina.** Ann. d'Ocul., 1931, v. 168, Jan., pp. 1-29.

The author divides his method into seven steps:

(1) Preliminary examination.

(2) Localization of retinal tears, including an accurate drawing. The tear is localized clockwise and in relation to the ora serrata or the pigment line marking the peripheral insertion of the retina seen in most cases with maximum mydriasis.

(3) The globe is marked at the limbus and countermarked at the opposite side, thus giving the direction. The distance of the ora serrata from the limbus depends upon the size of the globe, astigmatism, and the meridian, being 7 mm. on the nasal side and 8 mm. on the temporal side in the average eye and greater in myopia. The author feels that if exact localization is not made it is preferable to err by cauterizing in the direction of the disc. This prevents spreading toward the center of the retina and does not obliterate the view if a second operation proves necessary. The distance from the limbus is calculated in the following way. One disc diameter represents 1.5 mm. If a tear is four disc diameters from the ora serrata the distance will be 6 mm., which must be added to the distance of the ora serrata from the limbus namely 8 mm. if on the temporal side, making a total of 14 mm.

(4) Preparation for operation includes antihemorrhagic measures, intestinal purge, and adjusting the patient's position so as to place the tear as low as possible.

(5) At operation, after using local anesthesia, a thread is passed through the limbal marks to indicate the direction. The sclera is exposed and marked with a compass. Muscle insertions are rarely disturbed. Sclerotomy is performed with a Graefe knife because it is more accurate and the subretinal fluid can be released. Immediately a cautery point is inserted and held in place about two seconds. The conjunctival sutures are tied and the directing thread removed. Both eyes are bandaged.

(6) Postoperative care includes strict rest for five days and light diet to prevent bowel movements. The eye is dressed after three days, and again the fifth day, instilling atropin to aid the fundus examination made at this time. The time in bed depends upon the case and the site of the tear. Usually by the tenth day a few steps may be taken. A careful drawing is then made and compared with the original.

(7) When necessary to reoperate or to complete obliteration, one waits about fifteen days, longer in old people, in myopic patients, or when the second operation is to be done at the same site.

(Diagrams which accompany the article make the calculations and the marking of the globe very clear.)

H. Rommel Hildreth.

Hoffmann, Rudolf. **Is there a true embolism of the central retinal artery?** Klin. M. f. Augenh., 1931, v. 86, March, p. 603.

A hitherto healthy man of twenty-four years, four weeks after tonsillitis, suddenly became blind in the left eye under the clinical aspect of embolism of the central retinal artery, which after four days changed to a partial embolism of the superior temporal branch. A roundish longitudinal yellowish-white body could be very clearly seen in the vessel and was taken for an embolus, although the artery was not entirely obstructed. A greyish area of the retina corresponding to this did not regain its function. The macula was intact, as its arteries branched off before the obstruction. Arteriosclerosis and obliterating endarteritis were excluded. The Wassermann was negative. In fifty-five percent of cases of embolism of the central retinal artery up to forty-five years a valvular disease of the heart existed. This could not be demonstrated in this present case, which shows that embolism of a retinal vessel may occur without it. *C. Zimmermann.*

Hoffmann, Wolfgang. **The action of roentgen rays on the retina.** Zeit. f. Augenh., 1931, v. 73, Feb., p. 214.

The action of roentgen rays on the eyes has been fairly well known sinc

Birch-Hirschfeld's fundamental investigation. Changes in the retina have been rarely observed, particularly since the introduction of high potential and intense filtration. The author studied histologically two eyes which had been enucleated after intensive radiation of the region for tumors. Although the two cases had nothing in common except the therapy, the retinal lesions were identical, hence it seemed probable that the retinal lesions resulted from the radiation.

The first changes are in the ganglion cells without involvement of the other retinal layers. The cells swell, the vessel bodies disintegrate, and the fibrillæ are forced to the periphery where they form clumps and finally break up. The nuclei shrink, and finally vacuoles appear in the cytoplasm. In severe forms other layers are involved, and the rods and cones may disintegrate. Usually an edema, particularly of the nerve fiber layer, accompanies these changes. Hemorrhages occur. In all cases the bloodvessels are contracted and often an appreciable hyperplasia of the media occurs. The size of dose necessary to produce these changes is discussed.

F. H. Haessler.

Kotlarevskaia, S. Z. **On the correlation between pigmentary retinitis and glaucoma.** Arch. Oftalmologii (Russian), 1931, v. 8, pt. 1-2, pp. 159-162.

In a typical case of pigmentary degeneration of the retina the author observed bilateral chronic simple glaucoma. In another case of pigmentary retinitis the patient had lost sight in one eye as the result of an attack of acute glaucoma. The author believes that the simultaneous occurrence of glaucoma and of pigmentary retinitis is not a coincidence, but that both conditions are due to some underlying uveal lesion. *M. Beigelman.*

Koyanagi, Y. **Further remarks on pathogenetic changes of the choroidal vessels in nephritic retinitis.** Klin. M. f. Augenh., 1931, v. 86, Feb., p. 145. (See Section 7, Uveal tract, sympathetic disease, and aqueous humor.)

Lijo Pavia. **Cholesterin crystals in the subretinal fluid in atypical detachment.** Arch. de Oft. Hisp.-Amer., 1930, v. 30, Nov., p. 577.

Birefringent crystals in the subretinal fluid form a puzzling and rare phenomenon. Still rarer has been the opportunity for their direct examination. In the case reported a retinal detachment resulted from traction of proliferations extending from a postneuritic atrophic papilla. The subretinal fluid contained intensely brilliant floating particles. Three c.c. of the fluid was extracted through a scleral puncture back of the equator, the fluid reformed, and three months later eight c.c. was again evacuated. The cholesterol content of the fluid was 0.22 percent, and the same percentage was found in the blood at the time of the second puncture, although the record had been 2.1 percent at the time of the first puncture. Fundus photographs as well as microphotographs of the crystals illustrate the article.

M. Davidson.

Lijo Pavia. **Hypertensive retinitis.** Rev. Otō-Neuro-Oft., 1931, v. 6, Feb., p. 62.

Beginning with the original report of Richard Bright concerning obstruction of the central artery by kidney complications, the author traces the various conceptions of the disorder to the present time. The term "retinitis" is not accurate in this relationship, as it is not an inflammatory but a degenerative process, and the author proposes the name "hypertensive retinopathy."

The familiar star figure at the macula is due to a deposit of lipoid substances arising from a disturbance of the retinal and choroidal circulations at that point. This same figure has been seen in lead poisoning, chorosis, and papillary edema with cerebral tumor, and from this it has been argued that the choroidal vessels of the posterior pole unite directly with those of the optic nerve.

After reviewing various phases of the subject, the article closes with several pages of stereoscopic photographs of fundi illustrating the topics covered.

A. G. Wilde.

Lijo Pavia. **Intraocular hernia of the vitreous.** Rev. Cubana de Oft. y Oto-Rino-Laringol., 1930, v. 3, Oct., p. 210.

By this term the author refers to an intrusion of vitreous into the anterior chamber, following either accidental or surgical trauma. The case reported occurred in a mason who twenty-five days previously had been struck in the left eye by a particle of mortar that had fallen about five meters. There was slight corneal edema, with numerous pigmented deposits on Descemet's. The anterior chamber was markedly deepened, and the slit-lamp showed numerous pigmented and other highly refractile particles which were taken to be cholesterin, and several irregularly disposed trabeculæ. The lens showed pigment particles on its anterior surface, and the lower half was moderately opaque.

Diagnosis of vitreous hernias depends on the combination of trauma followed by marked deepening of the anterior chamber, the pigmented particles disseminated throughout, and the frequency of cholesterin crystals. The clearness of detail is slightly lessened, and the outline of the vitreous mass can be seen by means of the slit-lamp.

A. G. Wilde.

Mureddu, G. **On angiospasm of the retinal vessels.** Ann. di Ottal., 1931, v. 50, Feb., p. 164, and March, p. 278.

Graefe in 1859 was the first to demonstrate clinically an embolism of the central artery of the retina. Two years later he presented a typical picture of transitory blindness due to spasm of the retinal artery. Recent studies have shown that spasm of the retinal artery is accompanied by a construction of the capillary vessels with changes in the fundus and more especially in the macular region. Ocular endoscopy, known for a century though but recently applied clinically, shows that a form of capillary cramp begins in the region of the fovea and extends to the branches of the retinal arteries. Distinctions are made as to the intensity of the spasm, its seat of origin, as well as the clinic picture and course of the attack. In capillary spasm the macular region is affected, vision usually being reduced to not more than 5/10, and limited to the direct visual area. The only fundus change is a slight macular edema. There is no permanent loss of sight except when minute macular hemorrhages occur which may leave a small visual reduction. In spasm of the central artery the distinctive symptoms are almost complete loss of sight in the affected eye, marked edema of the retina, and narrowing of some or all of the affected vessels. Sight will be temporarily lost in the whole field or in the quadrant involved, or if the spasm continues for a lengthy period the damage to essential structures may be permanent. Common to the two conditions are the temporary character of the attacks, the tendency to recurrences, and the presence of certain general conditions such as arteriosclerosis, syphilis, cardiopathy, and toxemia.

The most effective remedy for the control of these neurovascular spasms has been found to be acetylcholine, introduced into the therapy as an energetic vasodilator by Villaret and Baçonçon in 1926. *Park Lewis.*

Pallarés, Juan. **Disciform degeneration of the macula.** Klin. M. f. Augenh., 1931, v. 86, Feb., p. 201.

The right macula of a woman aged seventy-two years showed a large whitish-grey, partly yellowish, uneven patch, 2.5 disc diameters long and 2 wide, over which several small tortuous vessels crossed, mostly from the lower temporal retinal arteries, but ending in the lower temporal vein. Two old hemorrhagic black foci were seen on both sides of the patch, and the area between this and the temporal border of the disc was occupied by brownish-black foci, atrophic pigment epithelium, and sclerosis of the choroidal vessels. The left eye showed similar changes in their incipiency. There was a large central scotoma in the right eye.

C. Zimmermann.

P'an, T. H. **Blood vessel formation in the vitreous in cases of retinitis prolif-**

erans. Nat. Med. Jour. China, 1931, v. 17, Feb., p. 110.

The author reports three and cites sixteen cases of retinitis proliferans with new bloodvessel formation in the vitreous. In the three cases reported, all were males under twenty-five years of age who showed new bloodvessel formation in the vitreous, accompanied by exudates and hemorrhages in the fundus. There was no clinical evidence of tuberculosis, but two cases gave a positive Mantoux reaction. Wassermann was negative. No foci of infection were found.

In the sixteen cases cited, all were males with ages ranging from nineteen to forty-seven years. One case showed a positive Wassermann reaction. In ten out of eleven cases the tuberculin test was positive. *M. E. Marcove.*

Pavia, L. **Light reflexes from the internal limiting membrane of the retina.** Ann. d'Ocul., 1931, v. 168, April, pp. 281-288.

The fundus of an injured eye was studied photographically during healing with regard to light reflexes. Edema of the retina disturbs the normal relations. *H. Rommel Hildreth.*

Pelláthy, B. **Retinal detachment and congenital uveitis in three siblings.** Zeit. f. Augenh., 1931, v. 73, Feb., p. 249.

In a six-year-old girl with normal eyes at birth, signs of blindness had appeared at the age of three months. Her three-year-old blind sister was able to see until she was one and one-half years old, while their six-months-old brother had never shown signs of visual power. In all a retinal detachment and signs of uveitis were found. No etiological factor was found in the family history, the personal history, or in the physical examination.

F. H. Haessler.

Pines, N, **Retinitis nephritica or albuminurica.** Brit. Jour. Ophth., 1931, v. 15, March, p. 129.

This is the concluding installment of a long series of papers on hypertension, sclerosis of the retinal vessels, and the present subject. This contribution is summarized a follows:

(1) Edema of the retinal anterior layers is seen ophthalmoscopically as a fine veil covering the larger vessels. If fully developed, it leads to "wool-patches" in the retina.

(2) Retinal edema is usually combined with renal insufficiency of varying degree and decreases or increases approximately pari passu with the latter, but an invariably coexisting conditon is arterial hypertension. With normal blood pressure retinal edema means only deficient oxygenation of the retinal cells.

(3) Albuminuric retinitis is ophthalmoscopically different from the sclerotic form. The appearance of this retinal edema will therefore mean for the patient all the difference between benign and malignant nephrosclerosis; and its degree and protean character will be analogous to those of nephrosclerosis in general. From the point of view of the organism albuminuric retinitis always means an inefficient kidney, although biochemical tests may appear satisfactory.

(4) It is not yet proved that albuminuric retinitis is caused by disease of the kidney; both organs may suffer from the same toxin. But the retina is more readily affected than any other part of the central nervous system, and this may be explained by the peculiarities of its histological structure. If the histology of the kidney were to prove similar in this respect to that of the retina, it would explain the parallel disturbance of both organs.

(5) The kidney affection characterized by fully developed albuminuric retinitis is clinically different from any other form of nephritis, and to it alone the term of "true interstitial nephritis" ought to be applied. It is a fatal disease of comparatively short duration, probably connected with a parenteral infection of the body, and its prognosis is bad in spite of apparently good response to biochemical tests.

D. F. Harbridge.

Saint Martin, de. **The efficacy and the diagnostic importance of vasodilators, acetylcholine in particular, in angiospasm of the retina.** Ann. d'Ocul., 1931, v. 168, Feb., pp. 102-124.

The author describes three clinical types of this condition. In the first type the blindness occurs suddenly. The second, or retrobulbar, type is unilateral or attacks one eye after the other. The last type is slowly progressive and simulates optic atrophy. In some cases the symptoms pass away shortly leaving no damage, while in others they persist and fail to respond to vasodilators.

Eleven case reports are given with numerous visual field charts. Retrobulbar injections of atropin, angioxyl and acetylcholine were given, the last being most effective but not always tolerated. An extensive bibliography of the years 1928, 1929, and 1930 is included.

H. Rommel Hildreth.

Salit, P. W., and O'Brien, C. S. **The behavior of the vitreous under different hydrogen and hydroxyl ion concentrations: a study in vitro.** Arch. of Ophth., 1931, v. 5, June, pp. 903-917.

The work reported in this paper was undertaken in an attempt to develop a method for measuring the degree of turgescence and deturgescence of the vitreous under varying hydrogen and hydroxyl ion concentrations in vitro, and also to apply this method for determination of such variations in the volume of the vitreous. The vitreous gel, when dialyzed against large amounts of salt-free hydrochloric acid and sodium hydroxide of various concentrations within a pH range of 1.0 to 9.0, absorbs or loses water with increasing concentrations of the acid or the alkali, respectively. The structure of the vitreous protects it somewhat against these changes, so that even when broken up by artificial means, only a part of the hydrogen or hydroxyl ions of the surrounding liquids were absorbed, unless the acid or the alkali was of such strength as to completely coagulate or dissolve the proteins. The presence of salts further retarded such absorption. The question as to whether certain types of glaucoma might be due to increase in alkalinity of the ocular humors is not, in the authors' opinion, answered by this work. It merely gives a more definite idea of the reaction of the vitreous, when in a relatively fresh and salt-free state, toward changes in the hydrogen and hydroxyl ion concentrations. It is normally in very sensitive balance. On one side there is extreme sensitiveness toward water, and on the other side there is its homogeneous, superfine structure plus a certain concentration of salts and other crystalloids. If this balance is frequently upset, defects will appear in the structure of the vitreous. A change in pH will result in changes in the structure, especially if the salts happen to be reduced in concentration. The cause for these changes in the vitreous may be removed by a counter reaction while the effect remains; thus, in glaucoma, we may find a normal pH, but have no proof that a change in pH may not have preceded the glaucoma.

The paper should be read carefully in its entirety. It is a most interesting study, accompanied by numerous interesting charts, but does not lend itself well to abstraction. *M. H. Post.*

Shoji, Y. **Three cases of retinitis with white spots treated successfully with A-O vaccine. Japanese cases of tuberculous retinitis.** Arch. d'Opht., 1931, v. 48, April, p. 241.

Three cases of bilateral retinitis in young Japanese individuals are reported in which the characteristic features of the disease were multiple white spots in the retina, hyperemia of the papilla, dilatation of the veins with hemorrhages, and perivascular proliferation of tissue. In no case was clouding of the vitreous or pathological change of the anterior segment noted. The patients became progressively worse under all treatment except the specific antigenic vaccine of Arina-Aoyama-Ohnawa, which produced immediate favorable results leading to

cure. Illustrations of the fundus pictures of the patients accompany the article and there is a discussion of the literature upon tuberculous retinitis.

M. F. Weymann.

Tille, H. **Unilateral instability of the retinal arterial tension in a syndrome of sympathetic hemiexcitation.** Ann. d'Ocul., 1931, v. 168, April, pp. 278-280.

A middle-aged woman had had crises of pain several times a day in the left side of the face for the last three years. Pilomotor reflexes on this side were hyperactive. Neurological examination was negative. The eyes were alike with equal vision and intraocular tension. The arterial tension of the left eye varied from one moment to the next and from day to day, whereas that of the right eye remained constant.

H. Rommel Hildreth.

Wölfflin, E. **Microscopic examination in a case of retinitis circinata combined with discoid degeneration of the macula.** Arch. f. Augenh., 1931, v. 104, May, pp. 47-59.

Wölfflin describes his pathological findings in a case of retinitis circinata combined with a discoid degeneration of the macula. It was possible microscopically to distinguish between the two conditions. The principal changes of retinitis circinata were cystic cavities in the region of the outer nuclear layer of the retina. These cystic areas were filled with a mixture of fibrin and hyalin. The cavities occurred in single rows as a rule; some areas, however, showed a layer of these cysts, those nearest the vitreous apparently containing a fluid substance. These findings correspond very closely to those reported by Elschnig, Behr, and Seefelder. *Frederick C. Cordes.*

Zur Nedden. **Detachment of the retina subsequent to accident.** Klin. M. f. Augenh., 1931, v. 86, April, p. 496.

Zur Nedden thinks that the importance of a tear in detachment of the retina is overestimated. If it were the cause the subretinal fluid would have to be identical with the vitreous. In the author's opinion, the tear is not even a regular symptom of detachment, as there are distinct cases of spontaneous detachment without a rupture. The detachment is not a disease of itself but rather a symptom of a complicated pathological process in which the retina, the choroid, and the vitreous participate. It continuously progresses and generally terminates with cataract and iridocyclitis.

There are also wrong conceptions of the effect of Gonin's ignipuncture. A tear can never be closed by burning, as the glowing cautery dipped in water at once ceases to glow. Its effect is not caustic but simply thermic, favorably influencing the disturbed nutrition of the interior. As the cause of retinal detachment is still unknown, its connection with an indirect injury can only be judged from clinical experience. As shown by a simple experiment contusion of the head can only in a limited degree be propagated to the interior of the eye. Zur Nedden recognizes in myopes an etiological relationship of extraordinarily strenuous physical efforts in the stooping position. On the other hand he reports four cases of severe contusions of highly myopic eyeballs and eight cases of lighter contusions observed during the last two years without damage to the retina. The time of occurrence of detachment is also important as it can only be attributed to injury if it occurs immediately or a few days after. Hence few cases remain of a probable etiological connection between indirect injury and detachment of the retina.

C. Zimmermann.

11. OPTIC NERVE AND TOXIC AMBLYOPIAS

Benedict, W. L. **Cranial sinus thrombosis: ophthalmologic aspects.** Surgery, Gynec. and Obstet., 1931, v. 52, Feb., p. 464.

The ocular signs of thrombosis of the cavernous and lateral sinuses are reviewed. Examination of the fundi should be made on all patients with suppurative disease of the ear. Changes

in the discs may precede changes in temperature and other signs of thrombosis. Choked disc is present in from ten to twenty percent of the cases. If swelling is progressive, the prognosis is much worse. True optic neuritis is only a rare complication. Of the ten cases cited, only two had choking of the disc, and these ended fatally; the others recovered.

Thrombosis of the longitudinal sinus is usually brought on by tumor, abscess of the brain, or extension from other sinuses. From the observation of fifteen proven cases, no eye signs were found. In the absence of tumor of the brain or involvement of the cavernous sinus, choked disc has not been observed in this group of cases. *M. E. Marcove.*

Blatt, Nicolaus. **Paralysis of accommodation and pupillary disturbances after lead poisoning.** Klin. M. f. Augenh., 1931, v. 86, April, p. 482.

Despite the great variety of ocular affections which have been reported as due to lead poisoning, relatively little has been said of pupillary changes and especially of alterations in the mechanism of accommodation. Blatt carefully studied 320 clinical histories of lead poisoning, and found only twenty-one cases of pupillary changes and two cases of disturbance of accommodation. He reports the following case. A man, aged thirty-seven years, whose occupation was installing and welding lead pipes, suffered in 1927 from violent vertigo, colic, vomiting, obscurations of vision, photopsia, and inability to read or write for weeks. All these symptoms disappeared after discontinuation of his work. In October, 1929, he complained of total amaurosis for several minutes, flickering, colic, and vomiting, and his gums showed the blue line of lead poisoning. During temporary amaurotic attacks the retina was very anemic, the optic disc discolored, and the retinal arteries narrow. The pupils, each 5.5 mm. wide, reacted directly and consensually very sluggishly to light and convergence, and had the irregular appearance of a rounded triangle. During the blind attacks reaction was still more retarded. The near point was at 56 cm. instead of 20.2 cm., and the range of accommodation was 1.7 diopters, being a loss of 3.35 diopters. The patient read with a +2.50 sphere at 30 cm. He gave up his work entirely, was put to bed, and was treated with iodine. On November twenty-first his condition was greatly improved and one month later all symptoms had disappeared. There had been no recurrence up to September, 1930.

Blatt considers with Elschnig that the obscurations and photopsias in lead poisoning are consequences of spasms of retinal vessels. Endarteritis also occurs in the retinal arteries. If the amaurosis develops gradually and if changes in the fundus and in the optic nerve are present, loss of vision will be permanent. The usual temporary amaurosis is due to irritation of the cerebral vessels. A tabulated synopsis of twenty-two cases reveals thirteen cases of absolute immobility of the pupils with mydriasis, three of reflex immobility, six with pupils of normal size (all of normal shape except three), and three cases of anomalies of accommodation. These changes of pupils and accommodation may be directly due to the toxic influence of lead on the nervous substance or indirectly to hyalin degeneration, endarteritis, and perivasculitis of the vessels with their consecutive disturbances of function.

C. Zimmermann.

Krol, A. **On the etiology of optic nerve atrophies.** Arch. Oftalmologii (Russian), v. 6, pt. 1-2, pp. 163-172.

Among 42,980 patients who were examined from 1925 to 1929 inclusive, 404 or 0.94 percent were suffering from optic nerve atrophies. The prevalent causes of these atrophies were syphilis (37.4 percent), meningitis (11.1 percent), and trauma (9.8 percent). Of interest is the relatively high incidence of optic nerve atrophies following acute infectious diseases: malaria in 2.2 percent, recurrent fever in 1.2 percent, and typhoid fever in 0.95 percent. The author emphasizes the fact that among the 150 syphilitic optic nerve atrophies,

seventy-one were observed in cases which had had no antisyphilitic treatment, and in the remaining seventy-nine cases the treatment had been entirely inadequate. He concludes that, contrary to Fuchs's opinion, proper antisyphilitic therapy administered in due time is of distinct prophylactic value in optic nerve lesions. In the management of syphilitic optic nerve atrophies the author advocates vigorous specific treatment. In nineteen cases thus treated, ten responded with an improvement in the visual functions; in seven cases the progress of the atrophy was definitely controlled; and in only two cases was the result unsatisfactory. Throughout the report no distinction is made between postinflammatory and primary atrophies of the optic nerve. *M. Beigelman.*

Löhlein, W. **Cone-shaped disc with formation of "drusen."** Klin. M. f. Augenh., 1931, v. 86, April, p. 433.

Five cases are described in patients ranging from fourteen to fifty-two years of age. The beginning of the well known slow process was most likely in childhood. Nine of the ten eyes had normal vision; the tenth, afflicted with choroiditis, had 0.8. The oldest patient, a man, who probably had been affected for a long time, showed a moderate concentric limitation of the visual field for colors. *C. Zimmermann.*

Manes, Antonio J. **Treatment of tabetic optic atrophy.** Arch. de Oft. de Buenos Aires, 1931, v. 6, March, p. 142.

In tabetic optic atrophy it is unknown whether we are dealing with a lesion in the ganglion layer of the retina, or an involvement of the central nervous system that has a predilection for the optic nerve. Fuchs has demonstrated hemianopsia in this condition, but generally the visual field shows nothing characteristic. The atrophy may manifest itself first as a central scotoma, a hemianopsia, or concentric narrowing. The field for colors fades in greater proportion than the form field. The atrophic process when once begun tends to progress to total blindness, the patient then remaining many years without other demonstrable lesions or symptoms. In the beginning it is difficult to differentiate between tabetic optic atrophy and the descending degenerative type of optic neuritis that is associated with syphilis of the central nervous system. This last leads gradually into the classical dementia paralytica.

When optic atrophy is suspected, repeated examinations should be made of the visual fields with the same test objects and under the same illumination and environment. This creates an actual standard for comparison.

According to Igersheimer, clinical improvement may follow suboccipital injection of salvarsan dissolved in cerebrospinal fluid, along with insufflation of air. This is an attempt to produce an artificial irritation of the meninges, which may serve as a mordant in fixing the salvarsanized spinal fluid. It is worth remembering that the disappearance of clouding and luminous sensations in the eyes following any form of treatment does not signify improvement. The average duration of the atrophic process is usually two to three years. By the intralumbar and cisternal injections one hopes to break through the hematocephalic barrier that seems to prevent intravenous and intramuscular injections reaching the central nervous system.

The observed cases were divided into three general groups. In the first group were those with duration up to ten months, and with one eye retaining vision better than 1/10. When treated simultaneously with salvarsanized serum and intramuscular injections of bismuth, seven out of eighteen went on to complete blindness in from two to twenty-seven months. Seven remained stationary, while three showed some improvement both in central vision and fields.

In the second group were those whose vision ranged from 1/10 to counting fingers. Two patients showed an improvement up to 6/30 under treatment. In the third group with advanced visual loss, the Swift-Ellis technique

was combined with malarial therapy. In one the vision improved from 1/120 to 1/60, but in the remainder there was no response to therapy. The author refers favorably to the habit of sending these cases to the rhinologist, who opens the sphenoidal sinus and then combines salvarsan treatment with malarial therapy. In this condition it is always necessary to consider the possibility of cerebral lues and to have neurological consultations at intervals. The best weapon is one of prophylaxis with aggressive treatment at the time of the initial lesion. *A. G. Wilde.*

Margotta, G. **Clinical research on the ocular complications of alcoholism.** Ann. di Ottal., 1931, v. 59, Jan., p. 43.

Clinical studies were made of the visual apparatus in twenty-nine alcoholics. Few changes were discovered in the anterior part of the globe. In the deeper structures some cases showed redness of the papilla with clouding of the papillary margin, others with pallor of the temporal half of the disc. In a few cases the eyeground was apparently normal. In certain instances the vision was of full acuity, in others the sight was diminished. As to the visual field the majority showed limitation for both form and colors, particularly in the vertical meridian in either the superior or inferior portion. In no case was there either a central scotoma for colors or an absolute scotoma. The optic nerve and retina were the parts especially affected. The maculo-papillary bundle was not more involved than other structures in the pathological changes. (Bibliography.)

Park Lewis.

Mariotti, C., and Laschi, G. **On retrobulbar neuritis of tuberculous origin.** Arch. di Ottal., 1931, v. 38, Jan., p. 1.

The authors, after enumerating the causes of retrobulbar neuritis, mention multiple sclerosis, paranasal sinus infections, alcohol and nicotine intoxications, lues, arteriosclerosis, cerebral tumors, and encephalitis as the most prevalent. They state that retrobulbar optic nerve lesions are sometimes due to tuberculosis. The pathology may start from the uveal tract, from the nerve head, without any other intraocular lesion, or from primary ocular tuberculosis with or without papillary changes. Intracranial tumor is excluded when the blind spot and fields are found to be normal. *David Alperin.*

Pedley, F. G. **The effects of lead on the vision. A case of subhyaloid hemorrhage.** Jour. Indust. Hygiene, 1930, v. 12, Dec., p. 359.

The literature on the effects of lead on the vision is reviewed, and five cases with eye disorders are referred to. Two of these were cases of optic atrophy, one of neuroretinitis, one of retrobulbar neuritis, and one of subhyaloid hemorrhage. The last case is described in detail. A man thirty-two years of age, who had been treated for lead poisoning six months before, suddenly developed a severe pain over the left eye. The next morning, vision in this eye was only light perception. Examination showed a large subhyaloid hemorrhage. Six months later the vision was 20/30, and the fundus negative except for a few pigment spots. The author considers the hemorrhage as probably due to the effects of lead.

M. E. Marcove.

Russo-Frattasi, G. **Retrobulbar neuritis from affections of the posterior sinuses.** Ann. di Ottal., 1931, v. 59, Jan., p. 38.

The optic nerve in its passage through the posterior sinuses is peculiarly vulnerable to infective processes. On the external side of the nerve at its anterior third is the ganglion which gives off the ciliary nerves. It is important to recall this relationship in order that neuroparalytic keratitis be distinguished from the usual corneal inflammation due either to mechanical lesions or infections. The arachnoid cavity forms a cul-de-sac terminating not far from the posterior pole of the globe. The nerve is enclosed in a sheath, and retrobulbar neuritis is a secondary phenomenon. Acute sinusitis affects the optic nerve less frequently than does the

chronic. Two forms of chronic sinusitis are especially subject to acute exacerbations; the latent form often clinically overlooked and the caseous form corresponding to cholesteatoma of the middle ear. It is rare that a sinusitis recognized and effectively treated gives rise to grave complications. It is the cases that have been neglected or overlooked that are the most dangerous. (Bibliography.) *Park Lewis.*

Smith, W. B. **Low grade ethmoiditis as a cause of certain eye conditions.** California and Western Med., 1931, v. 34, March, p. 88.

According to the author, a certain number of cases of retrobulbar neuritis are caused by accessory sinus disease, usually ethmoiditis. Both the eye and nose condition are ordinarily unilateral. The transmission of the disease to the optic nerve is usually through the blood stream instead of by continuity or contiguity. All cases do not require operation, the indication for operation being the eye findings.

M. E. Marcove.

Stählin, Sophie. **Is there a hereditary optic atrophy distinct from Leber's disease?** Arch. f. Augenh., 1931, v. 104, May, pp. 222-263.

There is a wide variation in the findings of Leber's disease. Stählin, however, from her work and a careful review of the literature, concludes that there are two types of hereditary optic atrophy distinct from Leber's disease. There is a congenital type of familial optic atrophy which is probably of the recessive hereditary type. In this disease, blood relationship of the parents plays an important part. There is also the complicated optic atrophy described by Behr. In this disease there is an unusually early appearing bilateral optic atrophy which Behr classed as hereditary. In addition to the eyes, other parts of the nervous system are involved. The condition seems primarily to affect boys.

Frederick C. Cordes.

Walker, C. B. **Retrobulbar neuritis and multiple sclerosis, part 2.** California and Western Med., 1931, v. 34, Feb., p. 83.

The relationship between retrobulbar neuritis and multiple sclerosis is stressed. The author considers the neuritis an early sign of sclerosis. A retrobulbar neuritis of over three months' duration without improvement must be differentiated from chiasmal lesions and multiple sclerosis. A guarded prognosis must be given in nonpurulent cases of retrobular neuritis in which recovery takes place in three to six weeks. Time alone will differentiate this from multiple sclerosis. Only four percent of the author's cases of retrobulbar neuritis are due to acute purulent involvement of the sinuses. Two cases are reported and field studies reproduced. *M. E. Marcove.*

12. VISUAL TRACTS AND CENTERS

Arruga. **Treatment of scintillating scotoma.** Arch. de Oft. Hisp.-Amer., 1931, v. 31, Feb., p. 103

Luminal has been employed with uniformly good results. Daily small doses, increased or decreased gradually, depending on results, and continued for some time after cessation of attacks, combined with attention to bowels, eight to nine hours sleep daily, and liberal but condiment-free diet have permanently relieved seven cases.

M. Davidson.

Bourguet, J. **Should hypophyseal adenomas and cysts of the pouch of Rathke be considered as suprasellar or intrasellar tumors?** Ann. d'Ocul., 1931, v. 168, May, pp. 379-397.

The author believes that these tumors should be considered as intrasellar. The embryology is discussed and observations given with illustrations to support this contention.

H. Rommel Hildreth.

Craig, W. McK., and Lillie, W. I. **Chiasmal syndrome produced by chronic local arachnoiditis; report of eight cases.** Arch. of Ophth., 1930, v. 5, April, pp. 558-574.

This paper is written in substantiation of recent reports of chronic local

arachnoiditis in the region of the chiasm, resulting in symptoms which fall under the chiasmal syndrome of Cushing. Seven cases were operated upon, in all of which the essential lesion was a localized arachnoiditis, with special emphasis upon the condition about the optic chiasm. In one case only was there a gliosis about the aqueduct in addition to the picture common to the others. In one case there was cecocentral scotoma without great alteration of the extent of the fields. In one case there was marked hydrocephalus, in another diabetes, in a third acromegaly, and in a fourth extreme obesity. Two cases recovered; five cases failed to do so.

Following this series of cases, the authors discuss the symptomatology of the basofrontal group, noting that steady increase in the lesions is indicative of tumor formation, while a rapid onset followed by gradual recovery is more suggestive of inflammatory disturbance.

The eighth case is then presented, which represents meningoencephalitis, with involvement of the chiasm. This case showed a definite right basal frontal syndrome in addition. A description of the operation follows.

Finally, in summary, the points upon which a differential diagnosis may be made are emphasized, noting that in certain cases, however, it is possible to establish a preoperative diagnosis. In these cases, the authors feel justified, owing to the grave prognosis, in carrying out the operation. They conclude that chronic local arachnoiditis about the optic chiasm is a definite clinical entity, presenting a syndrome similar to that produced by tumor in this region, and that surgical exploration is justifiable, even in cases where preoperative diagnosis cannot absolutely be established. *M. H. Post.*

Deery, E. M. **Two cases of tumor of the posterior cranial fossa causing visual hallucinations.** Bull. Neurol. Inst., New York, 1931, v. 1, Jan., p. 97.

Two cases of brain tumor beneath the tentorium in which visual hallucinations were prominent features are described. In one case, the hallucinations ceased after removal of a dermoid cyst which pushed the cerebellum upward.

The author, in discussing the mechanism involved in the production of this symptom, states that the tumor is usually supratentorial and involves directly or indirectly some part of the optic pathway. In these cases, however, the bulging tentorium might cause local irritation to the occipital lobes, and resulting involvement of the optic pathway. *M. E. Marcove.*

Kniskern, E. L. and P. W. **Hysterical monocular amaurosis.** Jour. Amer. Med. Assoc, 1931, v. 96, June 27, p. 2196.

The authors report a case of monocular total blindness occurring suddenly, and responding to complete recovery after a short period of suggestion. *George H. Stine.*

Petrović and Tschemolossow. **Rhythmic angiospasms in the nuclei of the ocular nerves.** Klin. M. f. Augenh., 1931, v. 86, April, p. 491.

A girl, aged seventeen years, had been frightened ten years previously. Following this she had several fainting spells with apparently epileptic convulsions. From that time she had a rhythmic closing and opening of each eye alternately, continuing even during sleep. After many years she consulted a physician who cured the left eye by six electric treatments. A sister of her mother showed the same symptoms after a fainting spell on delivery of a child, but was cured in two years. The right eye of the patient presented a typical paresis of the third nerve. After a few seconds the upper lid was raised in jerks and the eyeball turned inward to the normal angle with excessive miosis, to return shortly to the phenomena of typical paresis of the third nerve. The first phase lasted fifteen seconds, the second phase forty seconds. The vision in the first phase was approximately from 0.2 to 0.3 during the short period of opening; in the second

phase with miosis the refraction changed from four to five diopters due to the accommodative spasm. The fundi were normal. The healing of the left eye by electricity indicates a functional cause. Of various drugs instilled into the conjunctival sac, results were obtained only from cocain which shortened the phase of closure and altered the rhythms of mydriasis and miosis.

The following explanation of the phenomena is given: By a constant rhythmic stimulant emanating from the cortex of the brain, spasms of the small arteries arise in the nucleus of the third nerve, whose fibers innervate the right eyelid and pupil. The nuclei lose their function by this transiently disturbed blood supply and become for a time rigid. This rhythmic lack of innervation of the right eye with the third nerve brings about the described symptoms. *C. Zimmermann.*

13. EYEBALL AND ORBIT

Cosmettatos, G. F. **Concerning the structure of the cerebral visual center in the case of congenital anophthalmia.** Arch. d'Opht., 1931, v. 48, April, p. 282.

Comparison of the visual cortex of a normal newborn child with that of one afflicted with anophthalmia disclosed that the layer of Gennari, which is supposed to have the primary function of vision, was equally developed in both brains. As sections of the orbit revealed no normal retina in the anophthalmic child, there can be no relation between the development of the visual cortex and that of the retina, as has previously been stated.

M. F. Weymann.

Pi, H. T. **Hydrophthalmos in relation to nevus.** Nat. Med. Jour., China, 1931, v. 17, Feb., p. 95.

The association of nevus, hydrophthalmos, and glaucoma is discussed at length, and a review of the literature is given. The author was able to find thirty-five reported cases, all but three of which were unilateral.

The case reported by Pi is that of a young woman who had an extensive vascular nevus of the skin of the left side of the head and part of the neck ever since birth. Both upper and lower lids were involved to the margin. A few small nodules were present at the external canthus. The bulbar conjunctiva was entirely involved with a network of tortuous and dilated capillaries around the limbus. The iris contained a number of nodules on the surface in the region of the lesser circle. The fundus was negative, except for the glaucomatous cup. The tension was 58 mm. (Schiøtz). The eyeball was moderately enlarged. There had been a convergent squint of the left eye since birth, and the vision had always been poorer than that of the right. The right eye was entirely normal, except for a refractive error. *M. E. Marcove.*

Reilly. Wm. A. **Hypertelorism.** Jour. Amer. Med. Assoc., 1931, v. 96, June 6, p. 1929.

Hypertelorism is a developmental congenital anomaly of the lesser wings of the sphenoid bone; they ossify early in uterine life and grow excessively, thus causing the typical widely separated eyes. The skull is brachycephalic. The three classical associated conditions are family tendency, orbital separation, and retarded mentality. Strabismus may be present.

Four additional cases in American literature are reported, bringing the total to seventeen. *George H. Stine.*

Rosenbaum, Morris. **Congenital anophthalmos with orbitopalpebral cyst.** Arch. of Ophth., 1931. v. 5, June, pp. 884-889.

The author first carefully reviews the literature with regard to anophthalmos, microphthalmos, and various cysts that form about the orbit. True anophthalmos is rare and it develops from a pouch of the first cerebral vesicle. If proper invagination does not occur, the fetal cleft fails to close and cyst-like sacs may be formed. Many of these cases show a rudimentary or microphthalmic eyeball, which frequently cannot be discovered except on serial section. Failure of development of these

ocular vesicles may be the result of heredity, of internal or external causes, or of intrauterine inflammations. Hippel is said to have collected sixty-three cases of bilateral and twenty-three cases of unilateral anophthalmos. The present paper includes a report of one more case, which was accompanied by the formation of cysts. The author ascribes this case to heredity, the parents being cousins. *M. H. Post.*

14. EYELIDS AND LACRIMAL APPARATUS

Alcaino, A. and Rodriguez, M. **A survey of 300 West operations.** Arch. de Oft. Hisp.-Amer., 1931, v. 31, Feb., p. 65.

The joint experiences of the ophthalmological and otorhinological services of the El Salvador Hospital (Santiago de Chile) are summarized as follows: Conservative measures (sounding and catheterization) have been practically abandoned. Extirpation with and without removal of the palpebral lacrimal gland is only rarely resorted to, in such cases as obstruction of both canaliculi or of the common duct, fibrosis of the sac, ozena of the upper nasal passages, and peripheral facial paralysis. All epiphoras and permanent obstructions are indications for the West operation. Its advantages are as follows: (1) anatomical accessibility, (2) rapidity of execution, requiring only fifteen to thirty minutes, (3) possibility of immediate decision as to temporary contraindications because of restriction of operative field and sinusitis, (4) no bandage required, (5) possibility of repetition if first attempt fails, (6) greater percentage of cures, and absence of visible scar.

Proper functioning and permeability of the puncta and canaliculi are a prerequisite for success. As long as the superior canaliculus functioned, good results were achieved. Ectasia of the sac, in which there may be periodic obstruction in spite of permeability, is an unfavorable factor, but twenty-one cases were cured. Special preoperative and postoperative care is needed in atrophic small sacs and acute or chronic pericystitis and ozena. The results in the latter are not good, but the ozena is favorably influenced as long as the lacrimal passage is patent. Sequels of pure traumas of lacrimal passages were in one case relieved by the West operation. In most cases, however, the trauma had only aggravated a preexisting tuberculous and luetic disease, and offered no encouragement. Preliminary turbinectomy, septum resection, and ethmoid exenteration are at times required to make the field of operation accessible. Men have harder bones, and the harder the bone the poorer the landmark. All bits of bone must be carefully removed during the operation. In one case the orbit was entered. The most painful part of the operation is the incision of the sac, and its elimination is facilitated by the injection into the sac of a five percent solution of cocaine. nasal tamponing is not practised, a gauze drain being found sufficient. Eighty percent of cures were immediately obtained. Failures were due to granulations of the nasal mucosa. Watchfulness and immediate cutting away and cauterization of the granulations aborted half of the failures into ultimate successes, making ninety percent of permanent cures. Hereditary and acquired lues, at the bottom of the great majority of polypoid degenerations of nasal mucosa, is responsible for most of the failures of the West operations, and should be treated simultaneously. Tuberculosis of the lids and conjunctiva and diabetes are other factors to be dealt with as influencing cicatrization. *M. Davidson.*

Blanco, T. **Two new blepharoplastic procedures.** Arch. de Oft. Hisp.-Amer., 1931, v. 31, Feb., p. 79.

One procedure, employed in a case of upper lid epithelioma originating in the meibomian glands of the outer third, consisted in utilization of the nonadherent skin to fashion a sliding triangular flap which was pulled down to replace the excised lid border, and gave an excellent cosmetic result. Blepharorrhaphy of the middle third of the lids prevented contracture of the

orbicularis and contributed to the good result.

The other procedure was employed in an epithelioma of the inner canthus involving the neighboring lid portions. A curved incision following the nasal orbital border, and two incisions from its ends to the respective lid borders, facilitated, after extirpation of the included growth, reconstruction of the inner canthus. To relieve the tension of the lids on the sutures an ample external canthoplasty was done which gave as a result an added centimeter to the lid borders. The excellent cosmetic results obtained are illustrated.

M. Davidson.

D'Amico, D. **Oriental palpebral button.** Ann. di Ottal., 1931, v. 59, April, p. 347.

This rare disease is purely oriental and tropical unless imported. It is variously known as oriental ulcer, the button of Aleppo, of Biskra, of Egypt, and of the Sahara, and by its more scientific designation as dermatitis ulcerosa circumscripta. It is called by some modern authors Leishmaniosis cutaneum externum. Three cases are reported from southern Italy. It is endemic in Sicilia as in Calabria. It begins as a small red spot and often at first escapes observation. The spot becomes hard and elevated, gradually increasing in size and developing into a round protuberance that is free from pain. It passes through several stages resulting in an ulcerative condition of long duration. Necrosis is followed by cicatrization. With the Giemsa stain are shown numerous Leishman bodies. It is thought to be transmitted by the pediculus and the cimex lectularius.

Park Lewis.

Del Duca, M. **Initial syphiloma at the internal canthus.** Saggi di Oftalmologia, 1929, v. 5, p. 212.

The author reports a case of primary luetic ulcer at the inner canthus in a female patient, aged twenty-four years. The ulcer resulted from the breaking down of a tumefaction of two weeks' duration following a local traumatic excoriation.

The case was of interest in that the secretions from the ulcer were negative for spirochetes and the blood Wassermann reaction was also negative. However, the histologic findings, the presence of infiltration in the neighboring lymph glands with absence of local tenderness, the typical luetic character of the ulceration, and the rapid healing with slight remaining radiating scar following antiluetic treatment did not permit of any doubt regarding the nature of the lesion. *F. M. Crage.*

Del Duca, M. **Lipiodol in the radiographic study of the lacrimal passages.** Riv. Oto-Neuro-Oft., 1930, v. 7, Sept.-Dec., pp. 421-447.

For his radiographic study of the normal and pathologic lacrimal passages the author irrigates these structures with lipiodol through the superior or the inferior lacrimal punctum. He emphasizes the importance of using lipiodol, which has an antiseptic action and is not irritating. He prefers the occipito-buccal position of the head, which gives a perfect view of the filled passages. This method shows the exact location of a stricture, affords a good view of the sac ectasias, and shows the exact location of tumors and cysts and their relation to the sac. Thus one is enabled to make a right diagnosis of the pathologic conditions of the lacrimal passages and to decide whether a medical or surgical treatment is to be followed. The author obtained the complete elimination of the lipiodol in from one half to two minutes in normal sacs, and from one half to several hours in the stenosis of these passages. In the examination of more than one hundred radiographies the elongated shapes of normal sacs were noted; these the author classifies as cylindric, rectangular and vermiform. The pathologic sacs show a globular shape and are classified as oval, reniform, and pear-shaped, the latter being found in cases of complete stenosis of the nasal canal. The stenosis usually takes place at the union of the sac with the nasal canal, and in

these cases we see the infundibuliform canal. If the stenosis takes place at different levels of the nasal canal we see the moniliform canal. Stenoses, however, also take place in the canaliculus or in the sac below the level of the orbicularis tendon. A bibliography and eighteen figures accompany the article.

Melchiore Lombardo.

Gallenga, R. **Nodular tuberculosis of the subcutaneous tissue of the lids.** Arch. di Ottal., 1930, v. 37, Oct.-Nov.-Dec., p. 385.

After citing a few similar cases published in the literature, the author describes a case of tuberculosis with a single nodule in the subcutaneous cellular tissue of the right upper lid, and with subsequent diffusion of the process to the palpebral conjunctiva. This is the first case in which the Bacillus tuberculosis was in evidence. The bacillus was of the bovine type. The case report is accompanied by a careful clinical, histopathological, and biological study. There is also a voluminous bibliography. *David Alperin.*

Gallenga, R. **On a palpebral reflex from stimulation of the superior laryngeal nerve.** Arch. di Ottal., 1930, v. 37, Oct.-Nov.-Dec., p. 410.

The author succeeded in establishing the theory that the winking of the lid following stimulation of the superior laryngeal nerve is a reflex phenomenon, and is verified in the healthy as well as in the decerebrated dog. Not being able to follow the course of the stimulus, the author considers the hypothesis of association fibers existing between the tenth and seventh cranial nerves. *David Alperin.*

Goldfeder, A. E. **A new method for preventing deformations of the lids after simple excision of the tarsus.** Klin. M. f. Augenh. 1931, v. 86, April, p. 460.

Kuhnt's simple tarsectomy has proved a very valuable procedure against trachomatous complications of cornea and lids. However, entropion and some disagreeable deformations of the lids may be encountered after this operation. These are undoubtedly caused by the loss of the firm skeleton of the lid. To avoid such complications Goldfeder has devised a new method of tarsectomy supplanting the tarsus by skin-free auricular cartilage. The principal indication is the presence of a thickened infiltrated tarsus with or without corneal complications. Through a horizontal incision from 4 to 5 mm. from the lid border, the conjunctiva is dissected from the tarsus with a lance-shaped knife, the tarsus incised from 0.5 to 1 mm. from the lid border, grasped with forceps, and dissected from the adherent tissues. A corresponding piece of cartilage is excised from the auricle and transplanted under the conjunctiva; the edges are pulled taut over it, and the eye bandaged for from forty-eight to seventy-two hours. The skin of the auricle is not sewed, but is secured by the bandage. In all seven cases the operation was successful.

C. Zimmermann.

15. TUMORS

Alexiades. **A case of primary sarcoma of the lacrimal caruncle and semilunar fold.** Arch. d'Opht., 1931, v. 48, May, p. 341.

A boy seven years of age was burned on the right eye by a lighted cigarette. One month later a small red growth about the size of a large hazelnut had involved the region of the caruncle and semilunar fold. There was no adherence of the growth to the globe. The tumor was dissected free and excised, the wound being closed by sutures. Microscopically the neoplasm was found to be a round-cell sarcoma, probably originating in the plica. There has been no recurrence after more than one year, and, with the exception of an operative injury of the internal rectus muscle, the eye is normal.

M. F. Weymann.

Bardanzellu, Tomaso. **An interesting case of angioma of the inferior nasal lid angle, the caruncle, and the semilunar fold.** Arch. di Ottal., 1931, v. 38, Feb., p. 68.

This case is reported because angi-

oma of the lid diffusing to the caruncle and plica semilunaris is very rare. The article contains an enumeration of various forms of angiomata, photographs, and anatomical cuts showing the vascular supply of the lids. It is also accompanied by a bibliography.

David Alperin.

Bruce, G. M. **Retinoblastoma: its recognition when the fundus oculi is obscured.** Arch. of Ophth., 1931, v. 5, June, pp. 890-891.

It is simple to make a diagnosis of retinoblastoma when the media are clear, but not so simple when the fundus has been obscured by changes in the anterior segment. It should be suspected, however, when there is pseudohypopion, glaucoma, buphthalmos, invasion of the iris by the tumor, shrinkage of the globe with or without perforation, and finally intraocular hemorrhage with or without blood staining of the cornea. One case is reported.

M. H. Post.

Cochran, A. H., Jr., and Robinson, G. Allen. **Multiple epidermoid carcinoma of eye and lid.** Arch. of Ophth., 1931, v. 5, June, pp. 936-942.

The case reported was a man twenty years of age, who was seen first January 30, 1928. There was an ulcer on the lower right eyelid. Biopsy showed this to be a squamous-cell epithelioma, malignant in type. The ulcer cleared after 400 mg. hours of radium, used over a period of three and one-half months. The patient returned August 30, 1930. The left eye at this time showed an ovoid mass, having the appearance of granulation tissue, along the limbus from nine to twelve o'clock. It extended over the cornea about one-half way to the center. On the conjunctiva of the right eye, opposite the previous defect in the lid, there was a small, papillomatous growth of conjunctiva. The diagnosis was squamous-cell epithelioma of the left eye; papilloma of the right eye, showing some signs of epitheliomatous change. After dissecting the growths radiotherapy was instituted with excellent results. The case is of interest because of the nature and multiplicity of the tumors and their response to treatment, because multiple primary malignant growths of one organ are uncommon and such growths involving two or more unrelated organs are rare, and because of the youth of the patient. A considerable discussion of these tumors follows.

M. H. Post.

Galeazzi, Cesare. **Clinical and histopathological contribution to plasmoma of the cornea.** Arch. di Ottal., 1931, v. 38, Feb., p. 49.

Pascheff in 1908 reported three cases of plasmoma of the tarsal conjunctiva and one of the cornea. These were the first to appear in the literature. The present report of a corneal case is accompanied by photographs. The site of the tumor is that characteristic for pannus trachomatosus, and Lagrange considers that there is an affinity between this inflammatory neoformation and plasmoma. Two distinct forms, diffuse limbal, and circumscribed, are mentioned.

David Alperin.

Goldstein, I., and Wexler, D. **Rosette formation in the eyes of irradiated human embryos.** Arch. of Ophth., 1931, v. 5, April, pp. 591-600.

The conception of Wintersteiner that the rosette, characteristic of neuroepithelioma or neurocytoma, is derived from the neuroepithelium of the retina has been almost universally accepted. The rosette formation is the result of the palisade arrangement of the neuroepithelium about the external limiting membrane as a lumen. Benign rosettes have been found usually in fetuses at from five to eight months, or in infants. They were first found, after x-ray treatment to induce abortion, in two fetuses of six and seven months, respectively. In 1916 Pagenstecher experimentally produced well-formed rosettes in the retinas of rabbit embryos subjected to roentgen ray treatment in utero.

The material reported is derived from five fetuses subjected to large doses of x-ray during the second month

of pregnancy. The first three ended at from three and a half to four months, the fourth at six and a half months, and the fifth at seven and a half months. The last two each lived for one day. In addition to the rosettes, there were found failure of fusion of the upper portion of the fetal cleft at the site of the disc, and faulty development of the ciliary processes and iris.

The author concludes that all these conditions are the result of radiation at this period of development. The ectoderm or, more specifically, the inner wall of the optic cup, is the portion involved. The mesodermal elements remain normal. The lenses were also normal, probably the result of their stage of development at the time of radiation.

M. H. Post.

Grinker, Roy R. **Gliomas of the retina, including the results of studies with silver impregnations.** Arch. of Ophth., 1931, v. 5, June, pp. 920-935.

In order to make a detailed study of cellular types, specific methods of impregnation with silver salts must be used. Only such methods will show the fine processes of the various cells. Dilute formaldehyde solution without alcohol must be used, and frozen sections are required. Much has already been learned concerning these tumors by the usual methods, but further advance must be made from the stains such as described by the author. The tumors may be classified as follows: (1) medullo-epitheliomas, that is, those arising from the primitive retinal and ciliary body epithelium; (2) the retinoblastomas, or retinocystomas, which arise from a hypothetic bipotential indifferent retinoblastoma; (3) the neuroepitheliomas, which arise from the primitive spongioblasts or neuroepithelium. A detailed description of these three types of tumor and their modifications is given.

The author also sums up the results of his study of a neuroepithelioma of the retina preserved in a ten percent solution of formaldehyde which was received through the courtesy of D. T. Vail, Jr. It was found by the author's method that there were not only primitive spongioblasts, but astroblasts and astrocytes, with spongioblasts or neuroepithelial cells the predominating type.

Tumors of pigmented epithelium have not been described. The author feels that a better understanding of these retinal gliomas can be obtained by comparing the cells found in them with those found in different stages of the normal retinal elements.

M. H. Post.

Gualdi, V. **Contribution to the histopathology of cornu cutaneum of lid.** Ann. di Ottal., 1931, v. 59, Jan., p. 79.

The histological examination of a palpebral cutaneous horn (senile) showed a marked infiltration of lymphocytes with plasma cells, almost total disappearance of elastic tissue, proliferation of scattered collagenous masses, alteration in form and structure of the spinous cells with tendency to their fusion into a semblance of giant cells, and marked hyperkeratosis and parakeratosis. The growth was looked upon as a precancerous stage of a dermal neoplasm of epithelial origin with an atypical evolution likely to develop into an epithelioma. Such growths should be removed along with a generous zone of the circumscribed area. (Bibliography and plates.)

Park Lewis.

Kissin, N. E. **On inflammatory and malignant new formations of the conjunctiva.** Arch. Oftalmologii (Russian), 1931, v. 8, pt. 1-2, pp. 45-55.

Two rare cases of tumorlike formation in the conjunctiva are reported. A young man aged twenty-two years had a peculiar mass of hard consistency in the tarsal conjunctiva of the left eye. Under the microscope this new formation was found to represent advanced amyloid degeneration in the upper eyelid. The interesting histopathologic feature in this case was considerable ossification of the degenerated tissues.

In another case a small tumor in the conjunctiva was observed in a woman

twenty-five years of age. A biopsy was performed, and a microscopic diagnosis of an inflammatory pseudotumor was made. The condition, however, rapidly progressed. The orbit became invaded, and in view of the clinically malignant aspects of the process, a complete orbital exenteration was resorted to. This time on histopathologic examination a round-cell sarcoma was diagnosed. Whether the inflammatory findings in the first specimen were reactive to the malignancy, or whether there was a coincidence of inflammatory and neoplastic processes remained undecided.

M. Beigelman.

Lugli, L. **A contribution to the study of epibulbar epithelioma.** Arch. di Ottal., 1930, v. 37, Oct.-Nov.-Dec., p. 416.

The case described is interesting from the viewpoint of the progress of the neoplasm, its histogenesis, and the degree of its diffusion. The radical extirpation of the bulb and surrounding tissue was found, after the histopathologic report, to have been completely justified. The article contains an extensive bibliography. *David Alperin.*

Moore, R. Foster. **Diffuse neurofibromatosis with proptosis.** Brit. Jour. Ophth., 1931, v. 15, May, p. 272.

These are the clinical details of one of the author's cases together with reference to three others. A male, aged thirty-three years, when first observed gave a history of a slight fall at the age of six months, following which the right eye gradually pushed forward. At the time of examination the eye was markedly displaced forward and downward. The finger could be easily placed back of the globe, and the optic nerve could be felt. The eyelids closed well over the globe. Vision equaled 6/9; the field and fundus were normal. X-ray pictures showed the orbit to be very much larger than its fellow. The two outstanding features in the case are the pulsatile nature of the proptosis, not expansile as characterized in aneurysm, and a brachycephalic skull. The patient had many large and small nodules distributed over the body. Eight illustrations accompany the paper. *D. F. Harbridge.*

Scardapane, F. **Angioma of the lacrimal gland.** Saggi di Oftalmologia, 1929, v. 5, p. 497.

Two cases of primary tumor of the orbital portion of the lacrimal gland are described by the author. The patients were four and nine months old respectively. Histologically the tissue presented the characteristics of angioma. The cases lacked clinical symptoms of diagnostic importance. Of greater interest is the rare occurrence of this form of tumor in the lacrimal gland. *F. M. Crage.*

Scheerer, R. **Granuloma telangiectodes of the region of the caruncle and the lacrimal sac.** Klin. M. f. Augenh., 1931, v. 86, April, p. 445.

Granuloma telangiectodes of the exterior of the eye has not previously been described. A woman, aged sixty-four years, had observed for two and a half months an easily bleeding nodule covering the caruncle and the lacrimal canaliculus. It was extirpated and showed angiomatous tissue. There was no relapse after six months. In the second case, a woman aged fifty-two years, with purulent dacryocystitis and obstruction of the lacrimal duct, the wall of the extirpated tear sac showed granulation tissue proliferating into the lumen, which gave rise to repeated hemorrhages. The histological structure of both granulomas is described in detail and illustrated.

C. Zimmermann.

Trettenero, A. **Pathogenic relationship of trauma to orbital sarcoma.** Ann. di Ottal., 1931, v. 59, March, p. 272. (See Section 16, Injuries.)

Truc, H., and Dejean, C. **Diffuse and progressive angioma of the orbit, semiologic and operative difficulties.** Arch. d'Opht., 1931, v. 48, April, p. 290.

In a girl nineteen years of age progressive increase of exophthalmos of the right eye had been noticed since

birth. The eye was forced upward and outward by a mass in the inferior internal angle of the orbit. Radiography showed no abnormality. Operation was done by incision along the brow and inner angle of the orbit. A large tumor mass was removed with difficulty because it was diffuse, involved the internal rectus muscle, and bled freely. Hemostasis was finally accomplished but with incomplete removal of the tumor tissue and permanent damage of the internal rectus muscle. The tumor was a simple angioma or telangiectasis. The differential diagnosis between angioma, cyst, and lipoma is difficult.

M. F. Weymann.

Zeeman, W. P. C. **Pigment patches alongside a sarcoma of the choroid.** Ann. d'Ocul., 1931, v. 168, May, pp. 337-340.

Two fundus photographs are shown with pigment patches alongside the disc, a sarcoma of the choroid being present some distance out in the periphery. A photomicrograph demonstrates the pigment cells lying free above the pigment epithelium but beneath the retina. Such a patch should encourage search for a sarcoma.

H. Rommel Hildreth.

16. INJURIES

Aulamo, Rauno. **Experiments on the influence of roentgen rays on the eyes of rabbits.** Klin. M. f. Augenh., 1931, v. 86, April, p. 473.

During weekly roentgen ray treatments of a patient affected with ocular tuberculosis Aulamo observed obstinate conjunctivitis, which healed only after several months' treatment. He therefore studied the effects of various doses of roentgen rays on the eyes of rabbits and found intense shrinkage and atrophy of the conjunctiva and changes of the central portion of the cornea. The cataract starts from the posterior portion of the lens and is probably due to affections of the ciliary body. It may become total in a short time. Roentgen ray doses, so small that no alterations caused by them can be observed, may produce cataract after several months. As similar changes may occur in human eyes, the author, cautioned by his experiments on animals, confined himself in one hundred and fifty cases of ocular tuberculosis to sufficiently small doses at long intervals.

C. Zimmermann.

Balbuena, Fernandez. **Mycosis following corneal injury by a chestnut burr.** Arch. de Oft. Hisp.-Amer., 1931, v. 31, Feb., p. 84.

Grave keratitis occasionally results after a chestnut burr injury. Hypopyon, corneal perforation, and iris prolapse are also found, although in one case the prickle had remained for two years in the corneal parenchyma with only a mild reaction. The inoculation of the evacuated hypopyon mass, which looks like the sequestrum of an aspergillus mycelium, in the anterior chamber of a rabbit's eye reproduced an identical condition within from twenty-four to forty-eight hours. Examination of the enucleated eye, stained by phosphomolybdic acid or ammonium molybdate and followed by Unna blue, as well as by chloride of gold and Gram's, showed a pure culture of a fungus in three cases. For further identification, cultures of the burr prickle are being made in Raulin's, Saboureaud's, and Pallacci's media, and will be followed by inoculation into rabbits' eyes.

M. Davidson.

Gallenga, Riccardo. **Observations on a case of electric ophthalmia.** Arch. di Ottal., 1930, v. 37, March-April, p. 97.

Besides enumerating the usual subjective symptoms, the author emphasizes the discovery of miosis, increased intraocular tension, and annular scotoma yielding to pilocarpin instillation. This is, as he justly claims, hard to understand. Corneal epithelial changes could be seen only with the biomicroscope.

David Alperin.

Haas. **A practical foreign body needle.** Klin. M. f. Augenh., 1931, v. 86, April, p. 515.

The needle is curved, which allows it to be inserted under the foreign body

in the cornea. Alypine is recommended for anesthesia instead of cocaine, which softens the eyeball and injures the epithelium. *C. Zimmermann.*

Hoffmann, Wolfgang. **The action of roentgen rays on the retina.** Zeit. f. Augenh., 1931, v. 73, Feb., p. 214. (See Section 10, Retina and vitreous.)

Kung, H. G. **Injury of the cornea caused by Chinese native gunpowder.** Nat. Med. Jour. China, 1931, v. 17, Feb., p. 115.

Two cases of corneal injury due to explosions of Chinese gunpowder are presented. These cases differ from the ordinary corneal injury due to gunpowder in that the Chinese gunpowder contains sulphide of arsenic which not only causes mechanical irritation, but also a great deal of chemical irritation. This produces a chronic iridocyclitis and is followed sometimes by more serious complications. The absorption of this irritating chemical being very slow, the inflammatory process is prolonged.
M. E. Marcove.

Müller, H. K. **Chalcosis of both eyes with partial retrogression of the chalcotic changes.** Klin. M. f. Augenh., 1931, v. 86, April, p. 453.

Ocular changes by intraocular foreign bodies containing copper depend on their size and copper content. In some cases an abscess develops which leads to rapid destruction of the eye, while in others the eye is chiefly damaged by gradually developing chalcosis. Müller reports the clinical history of a boy aged eight and one-half years who was seen two hours after his eyes had been injured by the explosion of a brass shell. Small intraocular pieces of brass produced chalcosis of the cornea, lens, and retina of each eye. The copper salts first settled in the lens, then on the cornea, and much later on the retina. The chalcosis of the left lens was subcapsular near the anterior surface, and extended into the parenchyma. A bluish tint was observed on the posterior surface. The retinas showed orange-colored confluent spots of reddish luster. After five and one-half years the chalcosis of both retinas had almost entirely subsided and the copper cataract of the right eye had completely disappeared. *C. Zimmermann.*

Papagno, M. **Penetration of cilium into the anterior chamber with consecutive posterior synechia.** Ann. di Ottal., 1931, v. 59, Feb., p. 159.

In a penetrating wound of the cornea it is always necessary to think of the possible penetration of a cilium. Often irritation will follow as from a foreign body. It should be promptly removed to avoid more serious consequences. (Bibliography.) *Park Lewis.*

Scotti, P. **Changes in the crystalline lens from ultraviolet rays.** Ann. di Ottal., 1930, v. 58, Nov., p. 963; 1931, v. 59, Jan., p. 19, and March, p. 230.

An extensive study was made on eyes of animals. Irradiation on enucleated eyes caused marked fluorescence of the nuclear zone such as follows actual opacity. These changes were more marked in the eyes of older animals. In living animals the effects varied according to the duration of the time of exposure to the ultraviolet rays. In short exposures there was fluorescence not followed by opacity. Longer periods of irradiation caused the formation of vacuoles of the posterior surface of the lens following the confluence of the fibers which appeared thicker and more tortuous. Later experiments showed changes in the epithelium similar to those found in senile cataract, such as wrinkling, intracellular vacuolization, hydrops of the protoplasm and destruction of the nucleus. Opacity of the lens is produced only after long fractionated exposure to the rays. The opacity appears some days after the irradiation and is chiefly in the capsular and subcapsular region where the epithelium is most affected. In the living animals the ciliary epithelium becomes hyperemic, congested, edematous, stratified, and vacuolized.

The author concludes that the action of the rays on the cellular epithelium is different from that causing opacity of the subcapsular strata; the former

being due to selective action, the latter to altered osmosis of the lens from excessive imbibition of aqueous in the zone of greatest destruction of the cellular epithelium. Little importance is attached to the changes, which are wholly reactive and congestive, in the cellular epithelium. It is difficult to deduce from either clinical observations or experimental research any effect of the solar rays on the production of senile cataract. (Bibliography and plates.) *Park Lewis.*

Trettenero, A. **Pathogenetic relationship of trauma to orbital sarcoma.** Ann. di Ottal., 1931, v. 59, March, p. 272.

One month before consulting the author, a three-year-old child had fallen on the ground striking the right orbital region on an apple which she had in her hand. The eye had been disturbed for three weeks but had been apparently cured by topical application. The sight then began to fail, and iridocyclitis developed with almost total opacity of the lens. The inflammatory condition finally disappeared, after which the traumatic cataract was removed. Recovery was normal. Some two weeks later the eye presented exophthalmos, with great distress, limited motion, increased tension, diminished vision, turbid vitreous, and an elevation of the retina in the superior and inferior internal quadrants. Enucleation was performed. The eyeball was found to be enlarged, especially in its posterior segment. On sectioning, a growth was found to occupy the lower posterior portion of the eyeball. This growth had pushed forward the iridolenticular system and had eroded but not perforated the sclera. It was of a reddish-brown color. Sectioning showed sarcoma with prevailing pigmented cells. There was no evidence of degenerative changes. In the author's opinion a preexistent neoplasm was hastened by the traumatism. (Bibliography.) *Park Lewis.*

Weve, H. **Ophthalmia due to caterpillar hairs. (Ophthalmia nodosa seu pseudotuberculosa).** Arch. f. Augenh., 1931, v. 104, May, pp. 192-221.

Weve reports two cases of a pseudotuberculous inflammation of the eye due to the caterpillar hairs of euproctis chrysorrhoea. The essential points were the same in each case and the final diagnosis was made with the slit-lamp. At first there was a sudden, quickly disappearing, acute conjunctivitis. This was accompanied by photophobia, pain, and considerable discharge. After the conjunctivitis had subsided there developed a deep keratitis with iritis. In addition to the deep infiltrate, the cornea showed superficial flecks similar to those found in herpes. Later a definite descemititis developed. The keratitis was more or less limited to a section, while the iritis was diffuse. There were no vitreous changes present. During this stage there were present in both the palpebral and bulbar areas of the conjunctiva numerous glistening "tubercles". The slit-lamp showed these to contain a fine caterpillar hair.

Weve was able to reproduce the disease in rabbits' eyes and to demonstrate that the caterpillar hairs actually invade the eye, even being found in the lens. The work is to be continued in apes' eyes. *Frederick C. Cordes.*

17. SYSTEMIC DISEASES AND PARASITES

Cecchetto, E. **The first case of Schistosoma mansoni appearing in Europe with grave ocular complications.** Ann. di Ottal., 1931, v. 59, Feb., p. 155.

This form of disease is almost wholly tropical, but as a result of continuous travel from the colonies in the Orient the eggs of the parasite may be found in any country. The Schistosoma bilharzia affects the bladder, the Schistosoma mansoni the intestinal tract. The forms of the worms are quite different as are their manifestations. The case described appeared at the clinic at Verona, but the patient had made frequent trips to Venezuela where the disease is common. There was no evidence of the worm having entered the eye. A sudden loss of vision was found to be due to an embolus of the inferior nasal branch of

the central artery of the retina. Repeated paracentesis of the anterior chamber with exhibition of emetin and later stibium resulted in complete recovery of sight. The eggs of the Schistosoma mansoni were recovered from the patient's feces. *Park Lewis.*

Goldstein, Isadore, and Wexler, David. **Niemann-Pick's disease with cherry-red spots in the macula: ocular pathology.** Arch. of Ophth., 1931, v. 5, May, pp. 704-716.

In this paper, the authors describe the microscopic findings in a case of Niemann-Pick's disease. An episcleral ganglion was noted, and also cherry-red spots in the macula. This disease was first described by Niemann in 1914, and several years later by Pick. It resembles Gaucher's disease, but varies from it in some respects. The histological examination showed degeneration of the ganglion cell layer of the retina, vacuolation of the cells of the nuclear layers of the retina, with pigmented vacuolated cells in the choroid and vacuolated histocytes about the scleral vessels and episclera, with some infiltration of the cells among the scleral fibers. A group of ganglion cells in the episclera closely connected with the ciliary nerves showed changes similar to those of the ganglion cells of the brain and elsewhere, that is, there was granular degeneration of the Nissl bodies, with swelling of the cell and vacuolation of the cytoplasm. In addition to the cherry-red color at the macula, the discs showed unusual yellow pigmentation, as did the entire fundus, probably the result of lipoid deposition. An interesting color plate of the fundus accompanies the article. *M. H. Post.*

Gourfein, D. **Two new ocular symptoms in oxycephaly or premature cranial synostoses and their pathogenesis.** Arch. d'Opht., 1931, v. 48, Feb., p. 112.

Four cases of the above type are reported in which double congenital ptosis was present. In two of these insufficiency or paralysis of convergence existed. These two symptoms are not considered as having been directly caused by the cranial deformities, but probably had a common etiological factor with the latter. *M. F. Weymann.*

Lourie, O. R. **Focal infection: an etiologic factor in diseases of the eye.** Arch. of Ophth., 1931, v. 5, June, pp. 893-902.

The author of this paper has been much impressed, since his arrival in America nine years ago, by the importance laid to focal infection as the cause of many diseases of various types. Throughout the paper he decries procedures now in vogue and takes up a consideration of the work of a number of investigators in this country, endeavoring to show that their conclusions are not sufficiently well substantiated by their case reports. He feels that in many instances the value derived from the extraction of a tooth is due to the blood letting incident thereto. He also calls attention to the gravity of dental extraction, as instanced by numerous serious complications following this seemingly simple procedure. The paper is a plea for conservatism. *M. H. Post.*

Pillat, A. **The demonstration of vitamin A paucity by means of bacterioscopy of the conjunctiva.** Zeit. f. Augenh., 1931, Feb., p. 245. (See Section 5, Conjunctiva)

Rosenberg, M. **Diabetes and the eye.** Zeit. f. Augenh., 1931, v. 73, Feb., p. 207.

This is an internist's lecture on the eye changes observed in diabetics. From the oculist's point of view it is wise to divide diabetes into two types, the severe diabetes of youth and the mild form of old age. The aged diabetic very frequently has severe arterial disease, and most of the eye complications are not caused by the diabetes but by the angiosclerosis. Paresis of accommodation which has always been recognized as an occasional complication occurs with great frequency after insulin is first used. It clears up without treatment after one or two weeks. Aside from disturbances of su-

gar and acetone-body metabolism, the diabetic has disturbances of salt and water metabolism, as yet not exhaustively investigated. This probably accounts for the harmless insulin edema as well as the changes of refraction which vary between plus 4.00 and minus 9.00 diopters. The myopia results from swelling of the lens. Whether hypermetropia is the result of lens shrinkage or merely becomes manifest as a result of paresis of accommodation is not known. Paralysis of the external muscles is rare.

The retinitis of diabetes is never found in young persons and is a vascular retinitis. However, the form of retinitis with white spots about the macula and disc is so characteristic that the glycemia or diabetic metabolism may be an accompanying etiologic factor. The hemorrhages have nothing to do with diabetes. Most so-called diabetic cataracts are senile cataracts in diabetics. Doubtless an untreated diabetic ages more rapidly. More difficult is a pathogenetic explanation of the rare bilateral cataract of the youthful diabetic. Many internists regard it as specific. Hypotony of the bulbus in diabetic coma is a frequent, though not constant, finding and is a manifestation of general dehydration. Though diabetics are less resistant to infiltrative and purulent infections, inflammatory lesions of the eye have not been more frequent in these than in other patients of the internists. Therapy is useless in the complications which are senile or vascular in origin, but is extremely helpful in infections and operations. *F. H. Haessler.*

Sédan, J. **Iritis and familial hemophilia.** Ann. d'Ocul., 1931, v. 168, April, pp. 264-271. (See Section 7, Uveal tract, sympathetic disease, and aqueous humor.)

Solares, Aniceto. **New observation concerning orbital hydatid.** Arch. d'Opht., 1931, v. 48, April, p. 298.

A woman forty-seven years of age first noticed swelling and pain in the right orbit. Twenty days later diplopia appeared and was followed by marked exophthalmos. A tumor mass, which was soft and nonadherent, was present at the superior internal angle of the orbit. Operation was done by incision through the conjunctiva of the cul-de-sac. The cyst was punctured and examination of its contents showed a scolex and hooklets. One percent formaldehyde was injected into the sac followed by removal of the cyst membrane. The extreme rapidity of the development of symptoms is noteworthy.

M. F. Weymann.

Vancea, P. **Is there an iris symptom complex characteristic of tabes and paresis?** Zeit. f. Augenh., 1931, v. 73, Feb., p. 254. (See Section 7, Uveal tract, sympathetic disease, and aqueous humor.)

Weve, H. **Congenital mesodermal dystrophy, Martan type.** Arch. f. Augenh., 1931, v. 104, May, pp. 1-46.

Weve describes the typical clinical picture of dystrophia mesodermalis congenita. The condition is much more prevalent than a review of the literature would indicate. It is hereditary and to some extent resembles amyotonia congenita. In the typical case, there is an elongation of the hands and feet with a thinning of the bones on x-ray examination. The muscles are poorly developed, but show no paresis or degeneration; there is very little subcutaneous fat present. Children with the disease resemble living skeletons. In from forty to fifty percent of these cases, there is a bilateral congenital luxation of the lens. This, depending on the position of the lens, produces either a high myopia, up to forty diopters, or a high degree of hyperopia.

The author feels the eye findings can also be explained on the basis of a mesodermal disturbance.

Frederick C. Cordes.

Yagues Garcia, J. **Ocular myiasis.** Rev. Cubana de Oft., Oto-Rino-Larin., 1930, v. 3, Nov., p. 299.

The case reported is that of a woman thirty-eight years of age, who, while passing a meat market had felt a buzz-

ing fly strike her right eye violently. Yagues Garcia saw her the next day when she complained of the sensation of a foreign body in the eye, with photophobia, burning, swelling, and lacrimation. All these symptoms had increased rapidly. A loupe showed the folds of the inferior cul-de-sac to contain nine or ten white, immobile, rod-shaped bodies, which were easily removed by a cotton applicator. No ulcerations of the conjunctiva remained. Instillation of cocaine with washes of saline solution and sublimate ointment completed the treatment. The eye rapidly subsided to normal. *A. G. Wilde.*

18. HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Coppez, H. **Concerning the necessary physical fitness for driving of automobiles.** Arch. d'Opht., 1931, v. 48, April, p. 254.

The writer believes that an eye examination of professional drivers of automobiles is desirable, but that the requirements should not be too strict. Ocular examination of nonprofessional private drivers is impractical and unnecessary. The insurance companies do not believe that many accidents are caused by imperfect vision of automobile drivers and it has not been shown that the ratio of accidents is less in those countries where physical examinations are made than in countries which have no such requirements. A much more efficacious method of reducing accidents than that of seeking out the physically unfit drivers would be to have police patrol the roads and arrest reckless drivers before the accidents occur. *M. F. Weymann.*

Jackson, E. **Preparation for ophthalmic practice.** Surgery Gynec. and Obstet., 1931, v. 52, Feb., p. 446.

In this article Jackson outlines the requirements for the proper preparation of those specializing in ophthalmology. In all seven medical schools offering a systematic postgraduate course, only fifty-five students are provided for each year. The number of residents in special eye hospitals may bring this to a total of one hundred. A conservative estimate of the number of older men who drop out of the service is two hundred a year.

The author stresses fundamental training in the basic subjects, followed by a clinical course either in an eye hospital or under instruction of a competent teacher. For those who are already engaged in ophthalmic practice, short intensive yearly courses are urged. *M. E. Marcove.*

Moore, Paul G. **Value of vision in the modern horse.** The Horse, 1931, v. 12, no. 2, p. 23.

The author points out that the changed status of the horse in the past thirty years from purely a beast of burden to a participant in team activity, such as polo and hunting, has made a more careful scrutiny of the horse's vision advisable. In order to avoid danger to riders it is wise to have an ophthalmoscopic examination made at the time of purchase.

Lawrence T. Post.

Sédan, J. **William Nicati, 1850-1931.** Ann. d'Ocul., 1931, v. 168, April, pp. 241-243.

A biography.

H. Rommel Hildreth.

NEWS ITEMS

News items in this issue were received from Dr. Dean S. Smith, La Crosse, Wis. News items should reach **Dr. Melville Black, 424 Metropolitan building, Denver,** by the twelfth of the month.

Deaths

Dr. Alexander Rovinsky, New Haven, Connecticut, aged sixty-one years, died June 22, from an operation on the prostate.

Dr. John A. L. Bradfield, La Crosse, Wisconsin, a fellow of the American College of Surgeons, and a former member of the American Academy of Ophthalmology and Otolaryngology, aged seventy years, passed away on March 25, while on a pleasure trip to Florida.

Miscellaneous

The New York Ophthalmic Hospital has received $25,000 by the will of Dr. John E. Davis.

Under the will of the late Otto M. Eidlitz the Manhattan Eye, Ear and Throat Hospital received $75,984.

The New York Institute for the Education of the Blind was left $67,805 by the will of the late Georgiana M. Amidon.

The United States estimates in its recent census returns that there are 63,489 blind persons in this country. This is at the rate of 517 per million. New Mexico shows the highest, and Wyoming the lowest ratio of blind.

The Philadelphia County Medical Society has received, through the will of the late Dr. Lawrence Webster Fox, his medical library, medical instruments, pictures and portraits of distinguished physicians and surgeons, and $2,000 for the purchase of medical books and journals.

The second annual Summer Graduate Course in Ophthalmology of the University of Rochester was held August third to seventh. There were fifty registrants from many states. Much credit should be given to Dr. John F. Gipner under whose direction a most comprehensive and excellent program was conducted by local and visiting ophthalmologists.

Dr. Lawrence T. Post addressed the Los Angeles Ophthalmological Society on the subject of ultraviolet light, on August 19.

The ninth annual summer graduate course in ophthalmology and otolaryngology, given under the auspices of the Colorado Ophthalmological and Otolaryngological Societies, was held in Denver, July 20 to August 1, 1931. There were forty-two registered for the course from twenty-nine different states. The invited lecturers on the ophthalmological program were: Drs. Francis H. Adler, Philadelphia, on physiology of vision; Cecil S. O'Brien, Iowa City, on regional anesthesia and akinesia, cataract extraction, and the occlusion test for heterophoria; and Harvey J. Howard, St. Louis, on bacteriology and parasitology of the eye. The local ophthalmologists who participated in the course were Drs. Bane, Black, Danielson, Finnoff, Jackson, Marcove, O'Rourke, Shields and Thygeson. During two days of the course the Colorado Congress of Ophthalmology and Otolaryngology was held, in which, in addition to those already mentioned, Drs. Luther C. Peter, Philadelphia, and Wm. H. Wilder, Chicago, took part. There were the usual mountain trip and banquet. Discussion of questions at round table luncheons was a daily feature. The National Board for Ophthalmic Examinations conducted an examination of nine candidates at the Clinic of the University of Colorado.

The following item was received from Dr. P. J. Waardenburg, Arnhem, Holland, and Dr. K. T. A. Halbertsma, Delft, Holland.

"Interest of physicians in the theories of heredity increases more and more. The possibility of an international organisation of physicians and medical students before the VI International Congress of Genetics August 24-31, 1932 in Ithaca, New York, occurred to us. Perhaps it will be possible to discuss this at the meeting of the German Society of Heredity, September 13-17, 1931, in München and in the Section of Eugenics, Heredity and Natural Selection of the V Section of the International Institution of Anthropology and the XV International Congress of Anthropology and Archaeology (prehistorical) in Paris, September 20-27, 1931. Therefore we urge our colleagues to visit these meetings."

Professor F. Terrien and colleagues announce their regular annual course in ophthalmic surgery to be given at the clinique Ophthalmologique De L'Hôtel-Dieu commencing Friday October twenty-third. The course comprises twelve subjects and occupies as many days. The charge for enrollment is three hundred francs.

Societies

Dr. Walter F. Hoffman, Seattle, was elected president of the Pacific Coast Oto-Ophthalmological Society at its meeting in Los Angeles last May.

Dr. John M. Wheeler, professor of ophthalmology in the Columbia University College of Physicians and Surgeons, was recently appointed to the board of directors of the National Society for the Prevention of Blindness.